COMPARISON OF HEMODYNAMIC, EMERGENCE AND RECOVERY CHARACTERISTICS OF SEVOFLURANE WITH DESFLURANE IN GENERAL ANESTHESIA.

A STUDY OF 60 CASES

DISSERTATION SUBMITTED FOR THE DEGREE OF

DOCTOR OF MEDICINE

BRANCH – X (ANAESTHESIOLOGY)

APRIL-2012

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI,

TAMILNADU

BONAFIDE CERTIFICATE

This is to certify that this dissertation entitled **"COMPARISON OF HEMODYNAMIC, EMERGENCE AND RECOVERY CHARACTERISTICS OF SEVOFLURANE WITH DESFLURANE IN GENERAL ANESTHESIA."** is a bonafide record work done by **Dr. D.LAKSHMI SUDHA** under my direct supervision and guidance, submitted to the Tamil Nadu Dr. M.G.R. Medical University in partial fulfilment of University regulation for MD, Branch X –Anaesthesiology.

PROF. Dr.T.THIRUNAVUKKARASU, M.D, D.A,

Director, i/c.

Institute Of Anaesthesiology,

Govt. Madurai Medical College & Hospital

Madurai.

DECLARATION

I **Dr.D.LAKSHMI SUDHA** solemnly declare that this dissertation titled **"COMPARISON OF HEMODYNAMIC, EMERGENCE AND RECOVERY CHARACTERISTICS OF SEVOFLURANE WITH DESFLURANE IN GENERAL ANESTHESIA. "** has been done by me. I also declare that this bonafide work or a part of this work was not submitted by me or any other for any award, degree, diploma to any other University or board either in India or abroad.

This is submitted to The Tamilnadu Dr. M. G. R. Medical University, Chennai, in partial fulfilment of the rules and regulation for the award of Doctor of Medicine degree Branch –X (Anaesthesiology) to be held in April 2012.

Place: Madurai Dr. D.LAKSHMI SUDHA

Date:

ACKNOWLEDGEMENT

I am greatly indebted to **Dr.T.THIRUNAVUKARASU. M.D., D.A,** Director in-charge and Head of the Institute of Anaesthesiology, Madurai Medical College, Madurai for his guidance and encouragement in preparing this dissertation.

My heartfelt thanks to **Dr.S.C.GANESH PRABU, M.D., D.A,** Professor of Anaesthesiology, Madurai Medical College, Madurai for his guidance in doing this work.

I also thank my Professors **Dr.R.SHANMUGAM M.D.,D.Ch.,** and **Dr.A.PARAMASIVAN, M.D.,D.A,** for their constant support and guidance in performing this study.

I also thank my Assistant Professor **Dr. C.VAIRAVARAJAN M.D,** for his constant support in conducting this study.

My profound thanks to **Dr.EDWIN JOE, MD.,** The Dean, Madurai Medical College and Government Rajaji Hospital, Madurai for permitting to utilize the clinical materials of this hospital in the completion of my dissertation.

I gratefully acknowledge the patients who gave their consent and co-operation for this study.

TABLE OF CONTENTS

INTRODUCTION

The introduction of general anesthetics into clinical practice over 150 years ago stands as one of the seminal innovations of medicine. This single discovery facilitated the development of modern surgery and spawned the speciality of anaesthesiology.

General anesthesia can broadly be defined as a drug-induced reversible depression of the central nervous system resulting in the loss of response to and perception of all external stimuli. General anesthesia is a dynamic balance between the level of hypnosis, analgesia, and stimulation. It is usually defined as a triad of amnesia, analgesia, and muscle relaxation.

Inhalation anesthetics are the most common drugs used for the provision of general anesthesia. Adding only a fraction of a volatile anesthetic to the inspired oxygen results in a state of unconsciousness and amnesia. When combined with intravenous adjuvants, opioids and benzodiazepines, a balanced anesthetic technique is achieved that results in analgesia, further sedation or hypnosis, and amnesia. The popularity of the inhaled anesthetics for surgical procedures is because of their ease of administration and the ability to reliably monitor their effects with both clinical signs and end-tidal concentrations.

Inhaled volatile anaesthetics remain the most widely used drugs for maintenance of general anesthesia because of their predictable intraoperative and recovery characteristics. Management of haemodynamic stability and early recovery is the most important part of a standardized balanced technique.

Rapid induction and recovery may lead to faster operating room turnover times, shorter recovery room stays, and earlier discharges to home.

Over the last 15 years, there has been an explosive growth in the trend to provide cost-effective care in the practice of medicine. Ambulatory surgery is an increasingly important part of that trend. Ambulatory surgery continues to grow and thrive such that the vast majority (65–70%) of all surgical procedures is performed on an outpatient basis. Expeditious recovery and shorter hospital stays are necessary to improve efficiency of an ambulatory facility and reduce health care costs. One of the major factors that determine the speed of recovery from anesthesia is the choice of anesthetic technique. Although local and regional anesthesia techniques are increasingly used in the ambulatory setting because they allow a more rapid recovery , general anesthesia is still the most common anesthetic technique. An ideal general anesthetic technique should provide smooth and rapid induction, optimal operating

conditions, and rapid recovery with minimal or no side effects. It is also beneficial if the anesthetic technique allows for fast tracking (i.e, transferring patients directly from the operating room to the phase II unit, thus bypassing the postanesthesia care unit [PACU]).

Inhaled anaesthetics allow rapid emergence from anaesthesia because of easy titrability with inherent neuromuscular blocking effects that make them more suitable for ambulatory anaesthesia. The availability of less soluble inhalation anaesthetics such as sevoflurane and desflurane made us rethink about the selection of volatile anaesthetics for outpatient surgical procedures. Given the low blood: gas partition coefficient of sevoflurane and desflurane, faster emergence from anaesthesia is expected compared to traditional inhalation anaesthetics.

The purpose of this study was to compare the sevoflurane and desflurane in terms of hemodynamic, emergence and recovery characteristics.

AIM OF THE STUDY

This study was undertaken with the aim of prospectively comparing the hemodynamic, emergence and recovery characteristics of sevoflurane with those of desflurane in general anesthesia.

HISTORY

Inhalational anesthetics date back to the dawn of anesthesia. **Humphry Davy** first observed the analgesic effects of **Nitrous oxide** long ago in 1800, and called it as **" laughing gas"**. Horace Wells in 1844 described the use of nitrous oxide to facilitate the extraction of a tooth.

Henry Hill Hickman, in 1824 demonstrated that anesthesia could be induced and surgical procedures carried out using **carbon dioxide**. He was the one who introduced the concept of anesthesia using an inhaled substance. The first recorded general anesthetic administered in humans was in 1842, when both **C W Long and W Clarke** successfully induced anesthesia using **diethyl ether**.

The first successful public demonstration of anesthesia was in 1846, when **W T G Morton** successfully induced anesthesia using **Ether** on 16 October at Massachusetts General Hospital.

Discovered in 1831, the use of **chloroform** in anesthesia is linked to **James Young Simpson**, who, in a wide-ranging study of organic compounds, found chloroform's efficacy on 4th November 1847. Its use spread quickly and gained royal approval in 1853 when **John Snow gave it to Queen Victoria during the birth of Prince Leopold.**

These two agents, along with nitrous oxide, remained the mainstays of anesthesia along with nitrous oxide for the next 80 years, until the introduction of **cyclopropane** in 1930 by **Lucas and Henderson** in Toronto. Other agents used around this time included divinyl ether, trichloroethylene, and ethyl chloride. **Lehmann** first described the general anesthetic effects of **trichloroethylene** in 1911.

In 1932, **Booth and Bixby** observed that the greatest potential for non-combustible anesthetic agents lay with organic fluoride compounds, because the substitution of fluoride for other halogens reduces the boiling point, increases the stability, and generally reduces the toxicity of gases. Fluroxene, the first fluorine-containing anesthetic agent introduced in 1950.

First of the truly modern anesthetics, **Halothane** was first synthesised in 1951 by **C W Suckling** and was first introduced into clinical practice by Michael Johnstone in Manchester in 1956. Research into fluorinated compounds continued and has led to the production of all the modern anesthetic agents.

Methoxyflurane introduced in 1960, was soon withdrawn from the market due to its nephrotoxic potential. Fluroxene is explosive in concentrations greater than 3%.

Enflurane was produced by **R C Terrell** of Ohio Medical Products and it was first used in man in 1966. It is a halogenated methyl ethyl ether. **Isoflurane** was first produced in 1968 by **Dr R C Terrell**. It is a structural isomer of enflurane, a halogenated methyl ethyl ether.

Sevoflurane was first synthesised in the late 1960s at Baxter-Travenol laboratories by **R F Wallin** and co-workers. The first recorded use in humans was in 1981.

Desflurane was produced by **Dr Ross Terrell** and it was approved for clinical use in 1992. It is a fluorinated methyl ethyl ether.

INHALATIONAL ANESTHETICS

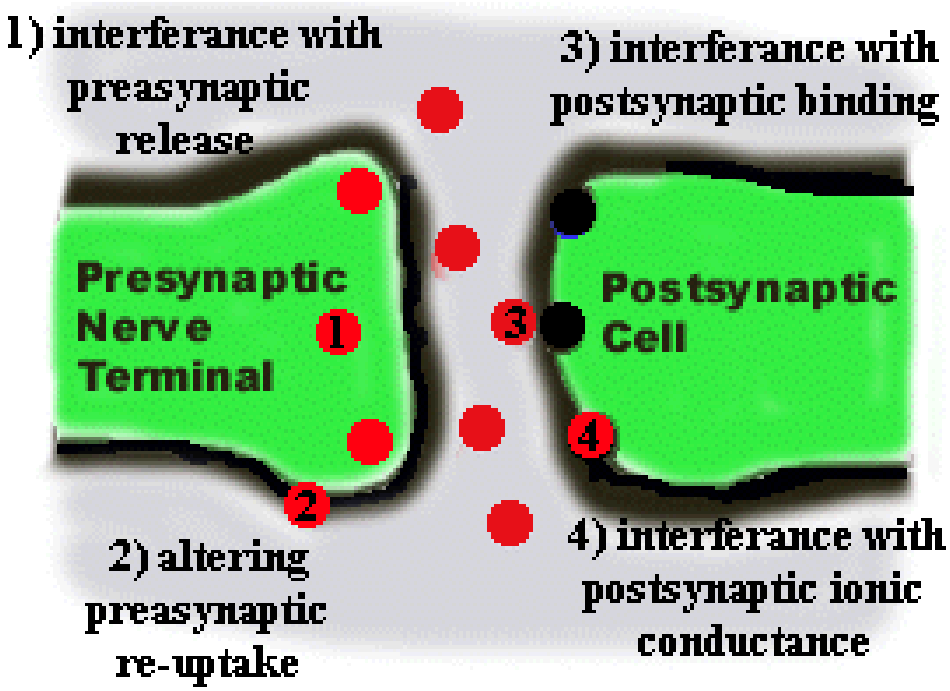
MECHANISM OF ACTION:

Inhaled anesthetics act in different ways at the level of the central nervous system. They may disrupt normal synaptic transmission by interfering with the release of neurotransmitters from presynaptic nerve terminal (enhance or depress excitatory or inhibitory transmission), by altering the re-uptake of neurotransmitters, by changing the binding of neurotransmitters to the post-synaptic receptor sites, or by influencing the ionic conductance change that follows activation of the post-synaptic receptor by neurotransmitters. Both, pre-synaptic and postsynaptic effects have been found.

Direct interaction with the neuronal plasma membrane is very likely, but indirect action via production of a second messenger also remains possible. The high correlation between lipid solubility and anesthetic potency suggests that the inhalational anesthetics have a hydrophobic site of action. Inhalation agents may bind to both membrane lipids and proteins. It is at this time not clear, which of the different theories is most likely to be the main mechanism of action of inhalation anesthetics.

The affinity of anesthetic drugs for lipids was soon recognized, which led to early lipid-based hypothesis of anesthetic action. The

MECHANISM OF ACTION



formulation of the **Meyer-Overton correlation** at the end of the 19th century was a watershed event, introducing a rigorous scientific approach into anesthesia research. Meyer concluded in 1899 that "All chemically indifferent substances that are soluble in fat are anesthetics … their relative potency as anesthetics will depend on their affinity to fat on the one hand and water on the other hand, that is, on the fat/water partition coefficient," a conclusion that was reached independently by Overton.

The Meyer-Overton theory describes the correlation between lipid solubility of inhaled anesthetics and Minimun Alveolar Concentration and suggests that anesthesia occurs when a sufficient number of inhalation anesthetic molecules dissolve in the lipid cell membrane. The Meyer-Overton rule postulates that the number of molecules dissolved in the lipid cell membrane and not the type of inhalation agent causes anesthesia. Combinations of different inhaled anesthetics may have additive effects at the level of the cell membrane.

However, the Meyer-Overton theory does not describe why anesthesia occurs. Mullins expanded the Meyer-Overton rule by adding the so-called Critical Volume Hypothesis. He stated that the absorption of anesthetic molecules could expand the volume of a hydrophobic region within the cell membrane and subsequently distort channels necessary for

sodium ion flux and the development of action potentials necessary for synaptic transmission. The fact that anesthesia occurs with significant increase in volume of hydrophobic solvents and is reversible by compressing the volume of the expanded hydrophobic region of the cell membrane supports Mullins Critical Volume Hypothesis.

A focus on protein targets gained momentum in the 1980s owing largely to the work of Franks and Lieb, who convincingly demonstrated that protein targets are also compatible with the Meyer-Overton correlation and proposed that anesthetics competitively antagonize protein function. The enantiomeric selectivity of several anesthetics further strengthened the case for specific binding sites on proteins, such as ion channels, as the principal molecular targets of inhaled anesthetics. Today, there is widespread (but not universal) acceptance of the notion that critical signalling proteins (e.g., ion channels and receptors) are the relevant molecular targets of anesthetic action even though the mechanisms of their modulation by anesthetics are debated.

MOLECULAR TARGETS OF INHALED ANESTHETICS:

Ion channels have emerged as the most promising molecular targets for inhaled anesthetics. Neurotransmitter-gated ion channels, in particular $GABA_A$, glycine, and NMDA-type glutamate receptors, are leading

candidates owing to their appropriate CNS distributions, essential physiologic roles in inhibitory and excitatory synaptic transmission, and sensitivities to clinically relevant concentrations of anesthetics. Other ion channels that are sensitive to inhaled anesthetics include the HCN (hyperpolarization-activated cyclic nucleotide-gated) family of channels that give rise to pacemaker currents and regulate dendritic excitability, two-pore domain (K_{2P}) "leak" K^+ channels that maintain resting membrane potential in many cells, and voltage-gated Na^+ and Ca^{2+} channels.

Inhaled anesthetics can be divided into two classes based on their distinct pharmacologic properties. The first class is the potent inhaled (volatile) anesthetics, which exhibit positive modulation of $GABA_A$ receptors, also produce significant, anesthesia-compatible effects on a number of other receptors or channels, including enhancement of inhibitory glycine receptors, inhibition of excitatory NMDA and neuronal nicotinic acetylcholine receptors, activation of K_{2P} channels, and inhibition of presynaptic Na^+ channels. Intravenous anesthetics such as propofol and etomidate represent more potent and specific positive modulators of $GABA_A$ receptors. The second class is the gaseous inhaled anesthetics, which include cyclopropane, nitrous oxide, and xenon. These anesthetics are inactive at $GABA_A$ receptors, but block NMDA receptors and activate

certain K_{2P} channels at clinical concentrations. The intravenous anesthetic ketamine is a more potent and specific blocker of NMDA receptors.

Each of the mentioned theories describes a unitary theory of narcosis. They all concentrate more or less on an unique site of action for inhaled anesthetics. The true mechanism of action of volatile anesthetics may be a combination of two or more such theories described as multisite action hypothesis.

MINIMUM ALVEOLAR CONCENTRATION (MAC):

Anesthetic potency of volatile anesthetics is measured by the minimum alveolar concentration (MAC). This value represents the minimum percent alveolar concentration of an anesthetic (at one atmosphere) that prevents movement in 50 percent of the subjects in response to supramaximal stimulus. A variety of noxious stimuli have been used to provoke response. For determination of MAC in humans, the usual stimulus used is surgical skin incision. In daily practice, MAC must be exceeded by a factor of 1.3 in order to assure sufficient surgical anesthesia for most of our patients. 1.3 times MAC will prevent movement in about 95 percent of the patients because MAC is ED50. The idea of measuring MAC is that after a short period of equilibration the alveolar concentration of the gas equals the blood concentration and a little later

equals the brain concentration. According to Dalton's law of partial pressure of gases, partial pressure can be substituted for concentration. Therefore , it represents after a short time the partial pressure of the anesthetic in the central nervous system (CNS) and it is therefore the most useful index of anesthetic potency.

MAC is age-dependent, being lowest in newborns, reaching a peak in infants, and then decreasing progressively with increasing age. MAC values for inhaled anesthetics are additive, which means that the addition of nitrous oxide will decrease the MAC of another volatile anesthetic. The MAC can also be reduced following administration of opioids. Inhalation anesthetics alone are not able to suppress hemodynamic responses to painful stimuli nor does MAC for skin incision predict the concentrations of inhalation anesthetics necessary to avoid the motor responses to other painful stimuli such as endotracheal intubation. As a rule of thumb, the addition of every one percent of alveolar nitrous oxide to another inhalation anesthetic will cause decrease in the MAC of that gas about one percent. Increases in MAC result from hyperthermia and hypernatremia. Decreases in MAC can result from hypothermia, hyponatremia, pregnancy, hypotension, and drugs such as lithium, lidocaine, opioids, and α2 agonists.

MAC creates a unifying principle of anesthetic depth for the inhaled anesthetics. Although each inhaled anesthetic has some pharmacologic peculiarities, in general they have parallel dose-response curves across drugs (e.g., isoflurane versus sevoflurane versus desflurane).

MAC- it is the end-tidal concentration of the inhaled anesthetic at one atmospheric pressure required to prevent movement in response to a noxious stimulus in 50% of subjects. (example - MAC of nitrous oxide-104%, sevoflurane-1.8 to 2 %).

MAC AWAKE - it is the end-tidal concentration associated with response to verbal stimulation in 50% of subjects. As a fraction of MAC, the MAC-awake of nitrous oxide (65% of MAC) exceeds that of desflurane, isoflurane, and sevoflurane (approximately 33% of MAC).

MAC BAR - the end-tidal concentration of the inhaled anesthetic that blocks adrenergic response in 50% of subjects.

UPTAKE AND DISTRIBUTION OF INHALED ANESTHETICS:

A series of partial pressure gradients, beginning at the vaporizer of the anesthetic machine, continuing in the anesthetic breathing circuit, the alveolar tree, blood, and tissue will ensure the forward movement of the gas. The principal objective of that movement is to achieve equal partial pressures on both sides of each single barrier. The **alveolar partial**

pressure governs the partial pressure of the anesthetic in all body tissues; they all will ultimately equal the alveolar partial pressure of the gas. After a short period of equilibration, the alveolar partial pressure of the gas equals the brain partial pressure. It is therefore most important to understand how to influence the alveolar partial pressure. It can be raised by increasing minute ventilation, setting a higher concentration on the vaporizer dial and by using a non-rebreathing circuit. Two special effects increasing the amount of gas in the alveoli have to be mentioned separately.

The concentration effect:

Describes how the concentration of the gas in the remaining alveolar volume can increase after some of the gas has been transferred into the blood.

The second gas effect:

Usually refers to nitrous oxide combined with an inhalation agent. Because nitrous oxide is not soluble in blood, its rapid absorption from alveoli causes an abrupt rise in the alveolar concentration of the other inhaled anesthetic.

All the above mentioned factors influence the inflow of gas into the alveoli.

Solubility, cardiac output, and the alveolar to arterial anesthetic gradient represent outflow factors. Inflow factors minus outflow factors equal alveolar partial pressure of the gas.

Solubility:

It describes the affinity of the gas for a medium such as blood or fat tissue. The blood/gas partition coefficient describes how the gas will partition itself between the two phases after equilibrium has been reached. Isoflurane for example has a blood/gas partition coefficient of 1.4. This means that if the gas is in equilibrium the concentration in blood will be 1.4 times higher than the concentration in the alveoli.

A higher blood gas partition coefficient means

- A higher uptake of the gas into the blood
- Slower induction time.
- It takes longer until the equilibrium with the brain partial pressure of the gas is reached.

Cardiac Output:

A higher **cardiac output** removes more volatile anesthetic from the alveoli and therefore lowers the alveolar partial pressure of the gas. The agent might be faster distributed within the body but the partial pressure in the arterial blood is lower. It will take longer for the gas to reach an

equilibrium between the alveoli and the brain. Therefore, a high cardiac output prolongs induction time.

Alveolar to venous partial pressure difference:

The **alveolar to arterial partial pressure difference** reflects tissue uptake of the inhaled anesthetic. A large difference is caused by increased uptake of the gas during the induction phase. This facilitates the diffusion of the gas from the alveoli into the blood.

Next we have to discuss the transfer of the gas from the arterial blood into the tissues such as the brain. It will depend on perfusion and solubility of the gas into different tissues. The brain/blood coefficient describes how the gas will partition itself between the two phases after equilibrium has been reached. Isoflurane for example has a brain/blood coefficient of 1.6 meaning that if the gas is in equilibrium the concentration in the brain will be 1.6 times higher than the concentration in the blood. All inhalation anesthetics have high fat/blood partition coefficients. This means that most of the gas will bind to fatty tissue as time goes by. The partial pressure of the gas in fatty tissue will rise very slowly. Inhalation anesthetics stored in such tissue in obese patients may delay awakening at the end of anesthesia. The body tissues have been divided into groups according to the level of perfusion and blood flow.

- Vessel rich group - VRG brain, heart, kidney, liver
- Muscle group - MG muscle and skin
- Fat group - FG large capacity / minimal flow
- Vessel poor group - VPG bone, cartilage and connective tissue

Tissue Group Characteristics

	Group			
	Vessel Rich	**Muscle**	**Fat**	**Vessel Poor**
Percentage of body mass	10	50	20	20
Perfusion as a percentage of cardiac output	75	19	6	0

Metabolism and elimination of inhaled anesthetics:

Metabolism is important for two reasons. First, intermediatory metabolites, end metabolites, or breakdown products from exposure to carbon dioxide absorbents may be toxic to the kidneys, liver, or reproductive organs. Second, the degree of metabolism may influence the rate of decrease in the alveolar partial pressure at the conclusion of the anesthetic.

Assessment of the magnitude of metabolism of the inhaled anesthetics is by

(a) Measurement of metabolites or

(b) Comparison of the total amount of anesthetic recovered in the exhaled gases with the amount taken up during administration. (Mass balance).

Determinants of metabolism:

The magnitude of metabolism of inhaled anesthetics is determined by the

- Chemical structure
- Hepatic enzyme activity
- Blood concentration of the anesthetic
- Genetic factors

Chemical Structure - the ether bond and carbon-halogen bond are the sites in the anesthetic molecule most susceptible to oxidative metabolism.

Hepatic Enzyme Activity - the activity of hepatic cytochrome P-450 enzymes responsive for metabolism of volatile anesthetics may be increased by a variety of drugs, including the anesthetics themselves. There is evidence in patients that brief (1hour) exposures during surgical stimulation increase hepatic microsomal enzyme activity independent of

the anesthetic drug or technique used. Conversely, surgery lasting more than 4 hours can lead to depressed microsomal enzyme activity.

Blood Concentration - the fraction of anesthetic that is metabolised on passing through the liver is influenced by the blood concentration of the anesthetic. For example, a 1 MAC concentration saturates hepatic enzymes and decreases the fraction of anesthetic that is removed during a single passage through the liver. Conversely, subanesthetic concentrations (< 0.1 MAC) undergo extensive metabolism on passage through the liver. Inhaled anesthetics that are not highly soluble in blood and tissues tend to be exhaled rapidly via the lungs at the conclusion of an anesthetic. As a result, less drug is available to pass through the liver continually at low blood concentrations conductive to metabolism. This is reflected in the magnitude of metabolism of these drugs.

Genetic factors - overall, genetic factors appear to be the most important determinant of drug-metabolising enzyme activity. In this regard, humans are active metabolizers of drugs compared to lower animal species such as the rat.

Induction and recovery from anesthesia with volatile anesthetics differ somewhat from each other. On induction all tissue partial pressures are zero. During recovery, different tissues in the body have a different

partial pressure of the inhaled anesthetic. Therefore, recovery is not as controllable as induction of anesthesia. In addition, increasing minute ventilation and concentration of the inspired anesthetic mixture can significantly accelerate induction. Increasing minute ventilation with high inspiratory oxygen concentration will increase the gradient of the inhaled anesthetic between pulmonary venous blood and the alveolar space and therefore increase the elimination of the gas. In summary, elimination of a volatile anesthetic depends on ventilation, cardiac output, and solubility of the gas in blood and tissue.

PHARMACOLOGY OF DRUGS

DESFLURANE

Desflurane was produced by Dr Ross Terrell. It was approved for clinical use in 1992.

Physical properties:

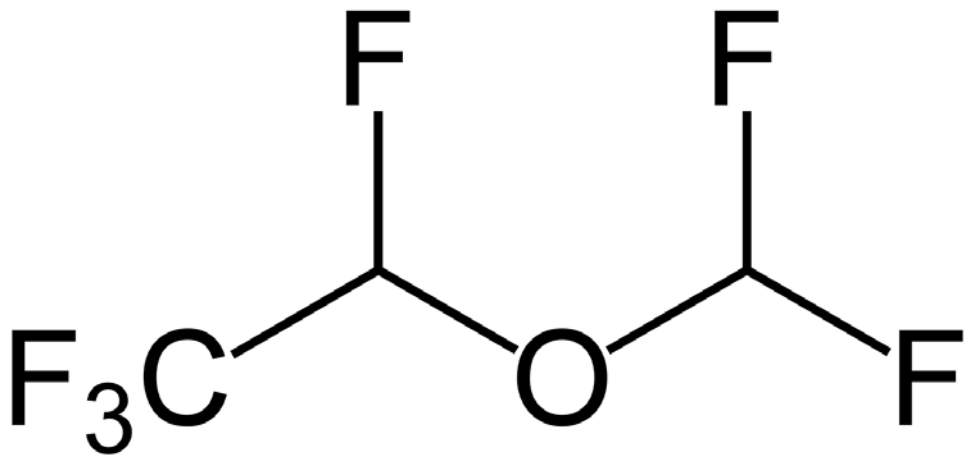
- Desflurane is a fluorinated methyl ethyl ether.
- Has a pungent odour, irritating and unpleasant to inhale.
- Molecular weight: 168.04 Dalton
- Boiling point: 22.8 ºC
- Saturated vapour pressure: 664 mmHg
- Blood-gas solubility: 0.42
- Minimum alveolar concentration: 6%

Chemical structure: 2,2,2-trifluoro-1-fluoroethyl-difluoromethyl ether

Pharmacokinetics:

Desflurane has the lowest blood-gas solubility of all the volatile anesthetics. Thus, it results in faster induction and faster recovery. Yasuda et al. have shown that equilibration of the FA/FI (fraction of alveolar to the inspired concentration) ratio of desflurane is higher than that of either isoflurane or halothane. At 30 min the FA/FI ratio of desflurane was 0.9 compared to 0.73 for isoflurane and 0.58 for halothane.

DESFLURANE: MOLECULAR STRUCTURE



Partition Coefficients of desflurane at 37ºC:

Blood-Gas : 0.42

Brain-blood : 1.3

Liver-Blood : 1.4

Kidney-Blood : 1.0

Muscle-Blood : 2.0

Fat-Blood : 27

The distribution of desflurane follows a five-compartment model which, it has been postulated, may be as follows – the lungs, the vessel-rich group of organs, muscle, fat around the vessel rich organs, and finally, peripheral fat.

The elimination of desflurane is almost exclusively through the lungs, with metabolism by the liver estimated to be less than 0.02%.

Pharmacodynamics:

Central nervous system:

Cerebral blood flow - desflurane has two effects on cerebral blood flow. Cerebral vasodilatation causes increase in cerebral blood flow while decrease in cerebral metabolic rate causes decrease in cerebral blood flow. Of these, vasodilatation predominates and there is global increase in cerebral blood flow.

Cerebral metabolic rate - desflurane causes a reduction in cerebral metabolic rate.

Intracranial pressure - drug induced vasodilatation tends to raise the intracranial pressure. This effect is not offset by hypocapnia.

The more rapid recovery associated with desflurane anesthesia may offer a small advantage in patients undergoing prolonged neurosurgical procedures.

Respiratory system:

Desflurane is irritating to the airway. So, it is unsuitable for inhalation induction. Concentrations of desflurane more than 6% cause coughing, breath-holding, and laryngospasm both in adults and children.

It is a potent respiratory depressant. It causes a dose-dependent decrease in tidal volume and an increase in respiratory rate, with an overall reduction in minute alveolar ventilation. Paco2 increases and the ventilatory response to carbon dioxide is reduced.

Desflurane causes concentration-dependent bronchodilation.

Cardiovascular system:

Desflurane causes peripheral vasodilatation resulting in dose-dependent tachycardia associated with depression in myocardial

contractility and a decrease in systemic vascular resistance. These changes occur at concentrations ranging from 0.83 to 1.66 MAC.

In ventilated patients, cardiac index remains unchanged and the systemic blood pressure falls. In spontaneously breathing patients, the cardiac index is increased.

It is a direct coronary vasodilator and produces an overall reduction in cardiac work. A rapid increase in the concentration of desflurane to greater than 1 MAC will cause an increase in heart rate and blood pressure.

Desflurane does not sensitize the myocardium to epinephrine.

Neuromuscular effects:

Desflurane depresses neuromuscular function. It can provide sufficient relaxation to allow tracheal intubation. It also potentiates the action of nondepolarizing muscle relaxants.

Toxicity:

Desflurane has low blood-gas and blood-tissue solubility and undergoes minimal metabolism less than 0.02 by liver. The metabolite, trifluoroacetic acid produced may interact with hepatic proteins and induce an immune response in susceptible patients.

There is no evidence of renal toxicity, even after prolonged exposure.

Desflurane, under certain conditions is degraded by carbon dioxide absorbers, and carbon monoxide is produced.

Desflurane is a trigger agent for malignant hyperthermia.

Clinical uses:

Desflurane has low blood-gas solubility and therefore provides most rapid induction and recovery. It undergoes minimal metabolism and the risk of toxicity is very low.

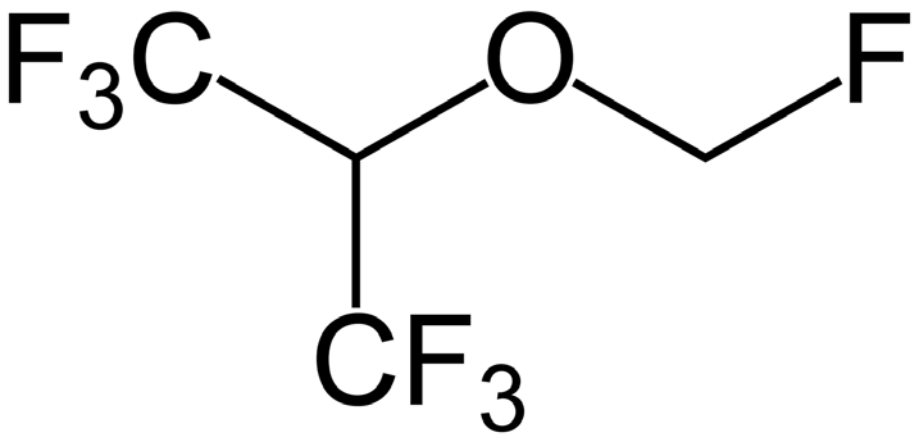
SEVOFLURANE

Sevoflurane was first synthesized in the late 1960s at Baxter-Travenol laboratories by R F Wallin and co-workers. The first published recorded use in humans was in 1981.

Physical properties:

- Sevoflurane is a fluorinated methyl isopropyl ether.
- It is colourless, non pungent to inhale, non-flammable and a liquid at room temperature.
- Molecular weight: 200.5 Dalton
- Boiling point: 58.5 ºC
- Saturated vapour pressure: 160 mmHg
- Blood-gas solubility: 0.69
- Minimum alveolar concentration: 2%

SEVOFLURANE: MOLECULAR STRUCTURE



Chemical structure: 1,1,1,3,3,3-hexafluoro-2-(fluoromethoxy)propane

Pharmacokinetics:

Sevoflurane has a low blood-gas partition coefficient which means that it has a rapid induction and rapid recovery. At 30 minutes after the start of anesthesia, the FA/FI(fraction of alveolar to the inspired concentration) for sevoflurane was 0.85 compared with 0.73 for isoflurane.

Partition Coefficients of sevoflurane at 37ºC:

Blood-Gas : 0.69

Brain-blood : 1.7

Liver-Blood : 1.8

Kidney-Blood : 1.2

Muscle-Blood : 3.1

Fat-Blood : 48

The distribution of sevoflurane follows a five compartment model.

Elimination of sevoflurane is primarily through the lungs and only a smaller amount is metabolised. The proportion metabolised has been estimated at between 1.6 % and 4.9%. Metabolism occurs in the liver, catalyzed by the cytochrome P450E1 enzyme.

Pharmacodynamics:

Central nervous system:

Cerebral blood flow: Sevoflurane causes cerebral vasodilatation and decrease in cerebral metabolic rate. Of these, vasodilatation predominates with a global increase in cerebral blood flow.

Cerebral metabolic rate: There is a reduction in cerebral metabolic rate.

Intracranial pressure: Sevoflurane causes a slight rise in the intracranial pressure. There is some evidence that sevoflurane has neuroexcitatory properties.

The cerebrovascular response to carbon dioxide and cerebrovascular autoregulation are both preserved under sevoflurane anesthesia.

Respiratory system:

Sevoflurane is pleasant to inhale and virtually has no irritant effect on the airway. This, combined with its low blood-gas solubility makes it suitable for inhalational induction. It is a respiratory depressant, causing a reduction in minute ventilation. Tidal volume is reduced. The ventilatory response to carbon dioxide is reduced.

Sevoflurane abolishes hypoxic pulmonary vasoconstriction in a dose-dependent manner in rabbit lungs. It is not proved in humans.

Sevoflurane is an effective bronchodilator.

Cardiovascular system:

Sevoflurane can produce direct myocardial depression through an action on calcium channels. It causes a dose dependent reduction in cardiac output and a reduction in systemic vascular resistance which results in a fall in systemic blood pressure.

It causes a reduction in pulmonary artery pressure that is not dose dependent.

Hepatic and renal blood flows are preserved.

Sevoflurane does not sensitize the myocardium to epinephrine. It is a coronary vasodilator. It does not cause the sympathetic-mediated cardiovascular stimulation associated with rapid increases in end-tidal concentration.

Neuromuscular effects:

Sevoflurane produces dose dependent muscle relaxation. At deeper levels of anesthesia, it facilitates tracheal intubation.

It potentiates the action of neuromuscular blocking agents.

Toxicity:

Metabolism is less significant because its tissue solubility is so low. Metabolism of sevoflurane results in production of inorganic fluoride and hexafluoroisopropanol. When sevoflurane is exposed to soda-lime or

baralyme, it is absorbed and degraded to a variety of compounds of which two are produced in significant amounts. They are fluoromethyl-2,2-difluoro-1-(trifluoromethyl) vinyl ether (Compound A) and fluoromethyl-2-methoxy-2,2-difluoro-1-(trifluoromethyl) ethyl ether (Compound B).

Toxic level of fluoride to produce nephrotoxicity is greater than 80μmol/litre. The lower the flow rate in the circle system, the higher will be the concentration of Compound A. The addition of water to soda-lime and the use of partially exhausted soda-lime seem to reduce the production of Compound A during low-flow anesthesia. While there is no evidence to date of serious long-tem renal injury associated with the use of sevoflurane, it would seem prudent to avoid prolonged exposure to sevoflurane with fresh gas flows of less than 2L/min.

Sevoflurane is not hepatotoxic. Sevoflurane triggers malignant hyperthermia.

Clinical use:

Sevoflurane has a low blood-gas solubility, so faster induction and faster recovery which along with its pleasant odour, makes it an ideal agent for inhalational induction. Toxicity has not been proved to be a clinical problem even with low flows for prolonged periods.

AMBULATORY SURGERY

Over the past 4 decades, ambulatory surgery has grown from less than 10% to over 70% of all elective surgical procedures. Ambulatory surgery has progressed from the practice of performing simple procedures on healthy outpatients to encompassing a broad spectrum of major procedures in outpatients with complex pre-existing medical conditions.

The availability of rapid, shorter-acting anesthetic, analgesic, and muscle relaxant drugs has clearly facilitated the recovery process after surgery. The range of acceptable ambulatory surgical procedures continues to expand, and patients are presenting for outpatient surgery with increasingly complex medical problems. As a result, anesthesiologists must play a more active role in the preoperative assessment and preparation of these patients. The role of the anesthesiologist has evolved from that of a physician primarily concerned with providing optimal surgical conditions and minimizing pain immediately after the operation to that of a perioperative physician responsible for ensuring that patients with coexisting medical conditions are optimally managed before, during, and after surgery. In modern ambulatory facilities, complex surgical procedures can be safely performed without sacrificing quality while minimizing use of hospital resources.

Perioperative Anesthetic Management for Facilitating a Fast-Track Recovery after Elective Ambulatory Surgery

Preoperative period- the coexisting diseases should be stabilised. Cessation of smoking should be encouraged. The anxiety and discomfort should be minimised. The patients should be adequately hydrated. Prophylactic measures to prevent postoperative complications like nausea, vomiting, pain, and ileus should be used.

Intraoperative period- Anesthetic techniques that optimize surgical conditions and also ensure a rapid recovery with minimal side effects should be used. Local analgesia via peripheral nerve blocks, wound infiltration, and/or instillation can be administered. Multimodal analgesia and antiemetic prophylaxis should be used. Avoid excessive fluid administration and minimise the use of nasogastric tubes and surgical drains.

Postoperative period- Adequate pain control should be provided. The patient who meets discharge criteria should be allowed to fast-tracked. Early ambulation and resumption of normal activities should be encouraged.

Duration of Surgery

The duration of surgery in the ambulatory setting was originally limited to procedures lasting less than 90 minutes because early investigators found that the operating and anesthesia times were strong predictors of postoperative complications (e.g., pain, emesis) and delayed discharge, as well as unanticipated admission to the hospital after ambulatory surgery. However, surgical procedures lasting 3 to 4 hours are now routinely performed on an ambulatory basis. Surgical procedures like dental extraction, facial fractures, excision of skin lesions, general surgical procedures, gynaecologic procedures, cataract excision, strabismus repair, arthroscopic procedures, closed reduction, hardware removal, otolaryngology procedures like adenoidectomy, laryngoscopy and mastoidectomy, pain clinic procedures like chemical sympathectomy, epidural injection and nerve blocks, plastic surgery procedures and urologic procedures are done under ambulatory surgery.

Patient Characteristics:

Originally, the majority of patients treated in ambulatory surgical facilities were classified as ASA physical status I or II. However, improved anesthesia and surgical care has allowed increasing numbers of medically stable ASA physical status III (and even some IV) patients to

undergo operations away from conventional medical centres. The risk of complications can be minimized if pre-existing medical conditions are stable for at least 3 months before the scheduled operation. Therefore, the ASA physical status should not be considered in isolation because the type of surgical procedure, the anesthetic technique, and a multitude of medical and social factors can also influence decisions regarding a patient's suitability for ambulatory surgery.

Age:

Age alone should not be considered a deterrent in the selection of patients for ambulatory surgery. Even the “elderly elderly” patient (>100 years) should not be denied ambulatory surgery solely on the basis of age.

Contraindications to Outpatient Surgery

Patients with the following conditions may be at an increased risk for postoperative complications and should be offered the option of overnight hospitalization:

- Potentially life-threatening chronic illnesses (e.g., brittle diabetes, unstable angina, symptomatic asthma)
- Morbid obesity complicated by symptomatic cardio respiratory problems (e.g., angina, asthma)

- Multiple chronic centrally active drug therapies (e.g., use of monoamine oxidase inhibitors such as pargyline and tranylcypromine) and/or active cocaine abuse
- Ex-premature infants less than 60 weeks' post conceptual age requiring general endotracheal anesthesia
- No responsible adult at home to care for the patient after surgery

REVIEW OF LITERATURE

1. Ravi jindal et el did a study on comparison of maintenance and emergence characteristics after desflurane or sevoflurane in outpatient anesthesia. They found that the time from administration of reversal agent to response to painful stimuli, to eye opening, to verbal commands and spontaneous eye opening were significantly shorter in patients administered desflurane than in patients given sevoflurane. For a given duration of anaesthesia, emergence from anaesthesia was significantly faster in desflurane compared to sevoflurane group. They concluded that both sevoflurane and desflurane provide a similar time to home readiness despite a faster wake up time with desflurane.
2. Earl M. Strum et al compared postoperative recovery after desflurane versus sevoflurane anesthesia in morbidly obese adults who underwent gastrointestinal bypass surgery via an open laparotomy. The times from discontinuation of volatile anesthetic administration to eye opening, squeezing hand, tracheal extubation and orientation were significantly shorter in patients given desflurane than in patients given sevoflurane. They concluded that morbidly obese adult patients who underwent major abdominal surgery in a prospective, randomised study awoke

significantly faster after desflurane than after sevoflurane anesthesia and the patients anesthetised with desflurane had higher oxygen saturation on entry to the Post Anesthesia Care Unit.

3. Wagih O. A. Ouda et al compared the emergence from anesthesia for day care surgery with spontaneous breathing using either desflurane or sevoflurane. They concluded that desflurane is associated with a faster emergence with no differences during the post-operative course except a somewhat higher incidence of airway irritation.
4. L. La Colla et al did a study to compare desflurane vs. sevoflurane kinetics and dynamics in morbidly obese patients and their recovery profile when no premedication had been used. Patients in the desflurane group reported early recovery times compared with those of sevoflurane group. They found that the time from discontinuation of the anesthetic drug to eye opening after verbal command, squeezing the observer's hand, extubation and ability to state their name and give their correct date of birth were all significantly shorter with a p value less than 0.001. The time from the discontinuation of the anesthetic drug to discharge from the recovery room was also significantly shorter in the desflurane group. ($p<0.001$). They concluded that desflurane

provides faster wash-in and wash-out than sevoflurane in morbidly obese patients, and recovery is much faster after desflurane administration when no premedication has been used.

5. M H Nathanson et al compared the recovery characteristics of desflurane and sevoflurane when used for maintenance of ambulatory anesthesia. They found that there were no differences between the two groups in the total doses of propofol, fentanyl and vecuronium used. Use of desflurane led to a more rapid emergence and shorter time to extubation when compared to sevoflurane. Recovery of cognitive function and discharge times were similar in the two groups. Thus they concluded that it would appear that sevoflurane is an acceptable alternative to desflurane for maintenance of outpatient anesthesia.
6. Rachel Eshima Mckay et al studied whether the airway reflexes return more rapidly after desflurane anesthesia than after sevoflurane anesthesia. They found that the time from stopping the anesthetic administration to appropriate response to command was longer in sevoflurane than desflurane. In addition, they found that the time from first response to command to ability to swallow 20ml of water without coughing or drooling was longer after sevoflurane. They concluded that

restoration of protective airway reflexes occurs significantly sooner after anesthesia with desflurane than after anesthesia with sevoflurane.

7. D Song et al did a study that was designed to test the hypothesis that using less soluble volatile anesthetics, desflurane and sevoflurane, as alternatives to propofol for maintenance of anesthesia facilitates the ability of outpatients to achieve postanesthesia care unit discharge criteria (i.e., fast-track eligibility) on arrival in the PACU after laparoscopic surgery. They found that compared with the propofol group, the times to awakening and to achieve a recovery score of 10 were significantly shorter and the percentage of patients judged fast-track eligible on arrival in the PACU was significantly higher, in the desflurane and sevoflurane groups. They concluded that, compared with propofol, the use of desflurane and sevoflurane for the maintenance of general anesthesia resulted in a higher percentage of patients being judged fast-track eligible after outpatient laparoscopic tubal ligation procedures.
8. Paul. F. White et al compared desflurane versus sevoflurane for maintenance of outpatient anesthesia and the effect on early versus late recovery and perioperative coughing. They found that although the

overall incidence of coughing during the perioperative period was higher in the desflurane group, the incidences of coughing during the actual administration of the volatile anesthetics did not differ between the two groups. Emergence from anesthesia was more rapid after desflurane; however all patients achieved fast-track recovery criteria before leaving the operating room. Finally, the time to discharge home and the percentage of patients able to resume normal activities on the first postoperative day did not differ significantly between the two anesthetic groups. They concluded that the use of desflurane for maintenance of anesthesia was associated with a faster emergence and a higher incidence of coughing. They found that despite the initial earlier recovery with desflurane, no significant differences were found between the two volatile anesthetics in the late recovery profile.

9. J Dupont et al studied the maintenance and recovery profiles after general anesthesia with sevoflurane, desflurane, and isoflurane in 100 patients undergoing pulmonary surgery. The anesthetics had comparable hemodynamic effects and arterial oxygenation during one-lung ventilation. They observed that emergence was twice more fast with desflurane than with sevoflurane or isoflurane. Early recovery was

also more rapid after desflurane. They concluded that in pulmonary surgery, desflurane, but not sevoflurane, allowed more rapid emergence and earlier recovery than isoflurane.

10. Pensado Castineiras A et al compared the anesthetic maintenance and early postoperative recovery and psychomotor function in patients who have been anesthetised with desflurane, sevoflurane or isoflurane during prolonged open urological surgery. They found that the recovery times in the operating room were significantly shorter after anesthesia with desflurane and sevoflurane than with isoflurane, with no significant differences between the desflurane and sevoflurane groups. They concluded that desflurane and sevoflurane demonstrated advantages over isoflurane during recovery from anesthesia in the operating theatre. No significant differences were found in psychomotor recovery, nausea and/or vomiting or requirements for postoperative analgesia.

11. Saros G B et al did a study on desflurane versus sevoflurane as the main inhaled anesthetic for spontaneous breathing via a laryngeal mask airway for varicose vein day surgery. They concluded that desflurane is associated with a faster emergence with no differences during the

postoperative course except a somewhat higher incidence of airway irritation.

12. Wellborn LG et al compared the emergence and recovery characteristics of sevoflurane, desflurane, and halothane in paediatric ambulatory patients. They found that emergence and recovery from anesthesia were significantly faster in the desflurane group compared with the sevoflurane and halothane groups. There was a significantly greater incidence of postoperative agitation and excitement in patients who received desflurane versus sevoflurane and halothane. There were no significant differences among the groups with respect to the time to meet home discharge criteria, in the time to drink oral fluids or in the incidence of postoperative vomiting. They concluded that , although desflurane resulted in the fastest early emergence from anesthesia, it was associated with a greater incidence of postoperative agitation. Sevoflurane resulted in similar emergence and recovery compared with halothane.

13. Ira Todd Cohen, MD et al examined the effect of a single intraoperative dose of fentanyl on emergence characteristics after desflurane or sevoflurane anesthesia in children undergoing

adenoidectomy. They found a similar incidence of severe emergence agitation after general anesthesia with desflurane and sevoflurane. Times to achieve extubation and postanesthesia care unit discharge criteria were shorter with desflurane than with sevoflurane. The emergence was faster with desflurane than sevoflurane by approximately three to five minutes. They concluded that desflurane allows for a more rapid emergence and recovery than sevoflurane. In children receiving desflurane or sevoflurane, the concurrent use of fentanyl in a dose of 2.5µg/kg resulted in a small incidence of emergence agitation.

14. Gupta A et al did a study on comparison of recovery profile after ambulatory anesthesia with propofol, isoflurane, sevoflurane and desflurane. They found no differences in early recovery between propofol and isoflurane. Early recovery was faster with desflurane compared with propofol and isoflurane and with sevoflurane compared with isoflurane. A minor difference was found in home readiness between sevoflurane and isoflurane but not among the other anesthetics. They concluded that comparing postoperative recovery after propofol, isoflurane, desflurane and sevoflurane-based anesthesia in adults

demonstrated the early recovery was faster in the desflurane and sevoflurane groups.

15. Arain SR et al did a randomized, prospective blinded study to determine the emergence profiles of desflurane and sevoflurane in morbidly obese patients when anesthetic drug titration was used. The demographic variables and hemodynamic parameters were comparable in both the groups. Hemodynamics, time to follow commands and to extubation, and results of Digit Symbol Substitution Test and Mini-Mental status Test did not differ between the anesthetic groups during recovery. They concluded that there were no differences in emergence and recovery profiles in morbidly obese patients receiving desflurane or sevoflurane when anesthetic concentration was carefully titrated.

MATERIALS AND METHODS

After institutional ethical committee approval, the study was conducted in 60 patients. All were ASA I and II patients undergoing elective surgical procedures under general anesthesia lasting for less than 3 hours but more than 1 hour duration. After getting consent, the anesthetic technique was performed.

SELECTION OF PATIENTS:

The patients selected for this study were of ASA Risk I&II undergoing elective surgical procedures under general anesthesia lasting for less than 3 hours but more than 1 hour duration. It was a prospective randomized controlled single blinded study.The patients exhibiting the following were excluded from the study.

- Significant cardiovascular, respiratory, hepatic, renal, neurologic, psychiatric or metabolic disease.
- Recent anesthetic exposure within previous seven days.
- History of allergic reaction to drugs.
- Potential susceptibility to malignant hyperthermia.
- Patient on chronic opioid analgesic or sedative treatment.

Age group:

Age of the patients ranged from 18 to 60 years.

PREOPERATIVE PREPARATION:

In the preoperative examination, all the patients were asked for any history of systemic illness like hypertension, diabetes, seizure disorder, bronchial asthma. History of any muscular dystrophies, neuromuscular disorders and family history of any malignant hyperthermia were noted. History of any allergic reactions to drugs and any chronic drug intake were noted. History of any previous surgeries was noted. Examination of the cardiovascular system and respiratory system were done. Assessment of the airway and the range of neck movements were done to rule out any difficult intubation. Apart from the routine blood investigations like haemoglobin, blood sugar, blood urea and serum creatinine, electrocardiogram and chest x-ray were ordered in patients greater than 40 years of age.

Hypertensive patients were advised to continue the antihypertensives on the day of surgery. Diabetic patients were advised to skip the morning dose of insulin. The surgeons were instructed to post the diabetic case first in the list and to send the patient to the operating room with the fasting blood sugar and urine acetone values taken on the day of surgery.

On arrival to the preoperative room, all patients were premedicated with injection midazolam 0.05mg/kg and injection glycopyrrolate 10µg/kg intravenously 30minutes prior to induction.

The patients were randomly allocated into two groups:

GROUP S- Sevoflurane 30 patients

GROUP D-Desflurane 30 patients

PROCEDURE DETAILS:

After shifting the patient inside the operating room, pre induction monitors pulseoximetry, non invasive blood pressure and electrocardiogram were connected. After securing the intravenous line and starting a crystalloid solution, all patients were induced with injection thiopentone sodium 5mg/kg, injection fentanyl 2µg/kg and intubated with injection succinylcholine 1.5mg/kg. After intubation capnography was connected. Group D was maintained with 3% desflurane and group S with 1% sevoflurane in 50% oxygen with 50% nitrous oxide. Neuromuscular blockade was maintained with injection vecuronium, initial bolus dose of 0.1mg/kg was given. Ventilation was controlled to maintain end-tidal carbon dioxide between 35and40mmhg. Injection fentanyl 0.5µg/kg was repeated every 30 minutes. Injection vecuronium 0.02mg/kg was repeated every 30 minutes. Mean arterial pressure and heart rate were noted before

induction and every 5minutes after induction. If there is any increase in the mean arterial pressure and heart rate more than 20% of the preinduction values, an additional dose of injection fentanyl 1µg/kg was given to maintain the hemodynamics. If there is any reduction in the mean arterial pressure more than 20% from the baseline value, it was treated with bolus of intravenous fluids and replacement of intraoperative blood loss. When the hemodynamics of the patient was unresponsive to the above measures, the patient was excluded from the study. Nitrous oxide and volatile anesthetic were discontinued after the last skin suture. Residual neuromuscular blockade was reversed with injection neostigmine 40µg/kg and injection glycopyrrolate 10µg/kg intravenously. Trachea was extubated when regular spontaneous breathing pattern was re-established and when the patients were able to open their eyes on command.

The time of discontinuation of anesthetic agents were noted as time zero for all the subsequent measurements and recovery times were determined at 1-minute intervals to awakening.

PARAMETERS OBSERVED:

- Number of additional doses of fentanyl needed.
- ***TIME TO***- 1. First spontaneous motion

  2. Response to painful pinch

3. Extubation

4. Recall of name

5. Hand grip

6. Achieve a PARS > 10 (post anesthesia recovery score of Aldrete and Kroulik)

This PARS records vital signs with patients receiving 0-18 points, that is 0-3 points for five physiological variables. One designated investigator administered all anesthesia; another assessed recovery.

PARAMETERS	**SCORE**
Consciousness	
Easily arousable, alert	3
Arousable, oriented, not alert	2
Arousable, not oriented	1
Not responding	0
Ventilation	
Normal	2
Not perfect, but requires no support	1
Airway requires support	0
Circulation (mean, supine, sitting)	
Arterial pressure difference	

< 10 %	2
10-20	1
>20 %	0

Horizontal nystagmus

Follow command, no nystagmus	2
Follow command, nystagmus	1
Fail to follow command	0

Countdown test (backward from 10 to 0)

Succeed right away	2
Succeed in 30 seconds	1
Fail in 30 seconds	0

STATISTICAL TOOLS

The information collected regarding all the selected cases were recorded in a Master Chart. Data analysis was done with the help of computer using **Epidemiological Information Package (EPI 2010)** developed by Centre for Disease Control, Atlanta.

Using this software range, frequencies, percentages, means, standard deviations, chi square and 'p' values were calculated. Kruskul Wallis chi-square test was used to test the significance of difference between quantitative variables and Yate's chi square test for qualitative variables. A 'p' value less than 0.05 is taken to denote significant relationship.

OBSERVATIONS AND RESULTS

Table 1: Age distribution

Age group	Desflurane group		Sevoflurane group	
	No	**%**	**No**	**%**
Up to 20 years	1	3.3	1	3.3
21-30 years	6	20	5	16.7
31-40 years	8	26.7	11	36.7
41-50 years	10	33.3	8	26.7
Above 50 years	5	16.7	5	16.7
Total	30	100	30	100
Range	19-57 years		20-60 years	
Mean	39.8 years		39.4 years	
SD	10.8 years		10.4 years	
'p'	0.7729 Not significant			

Cases studied in the desflurane group had an age of 39.8 $\pm$10.8 years and the sevoflurane group had an age of 39.4$\pm$ 10.4 years. There was no statistically significant difference.

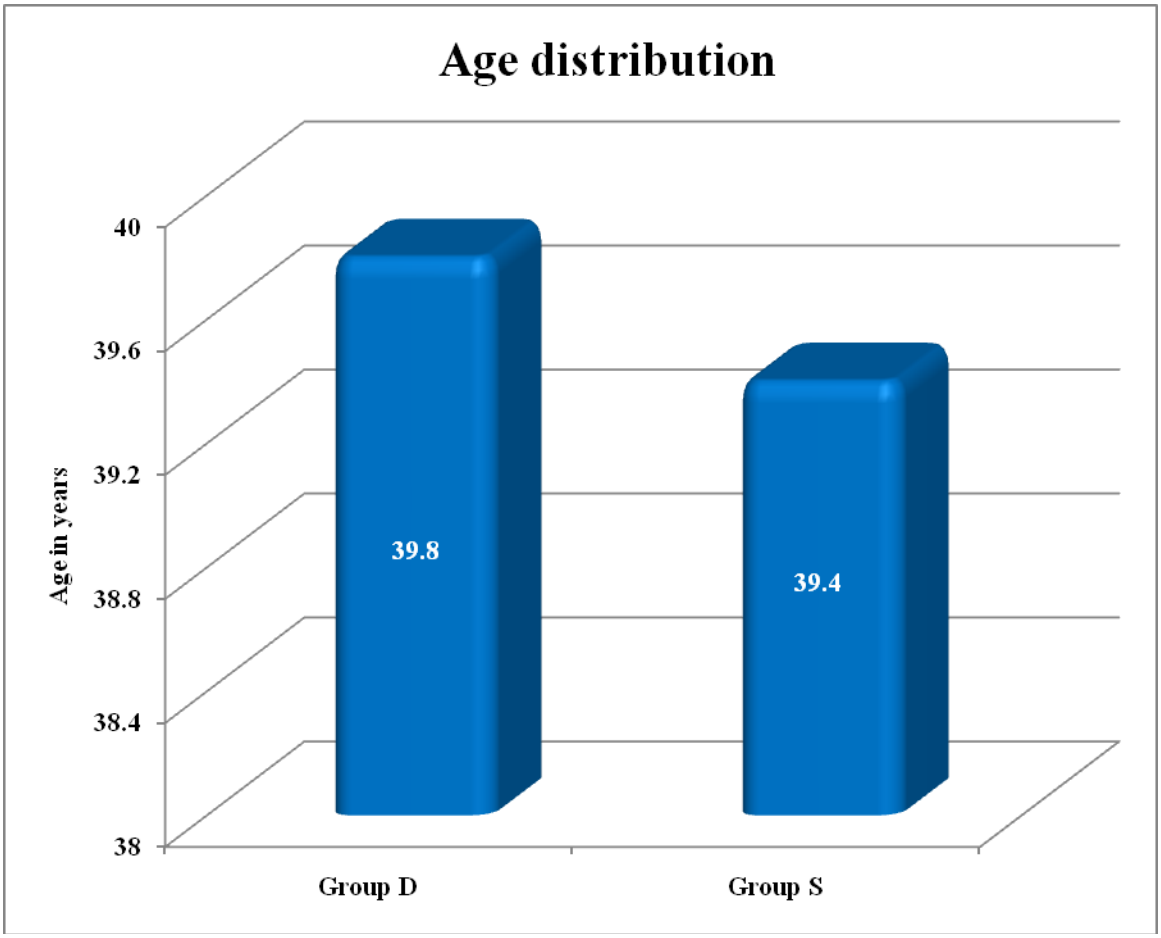
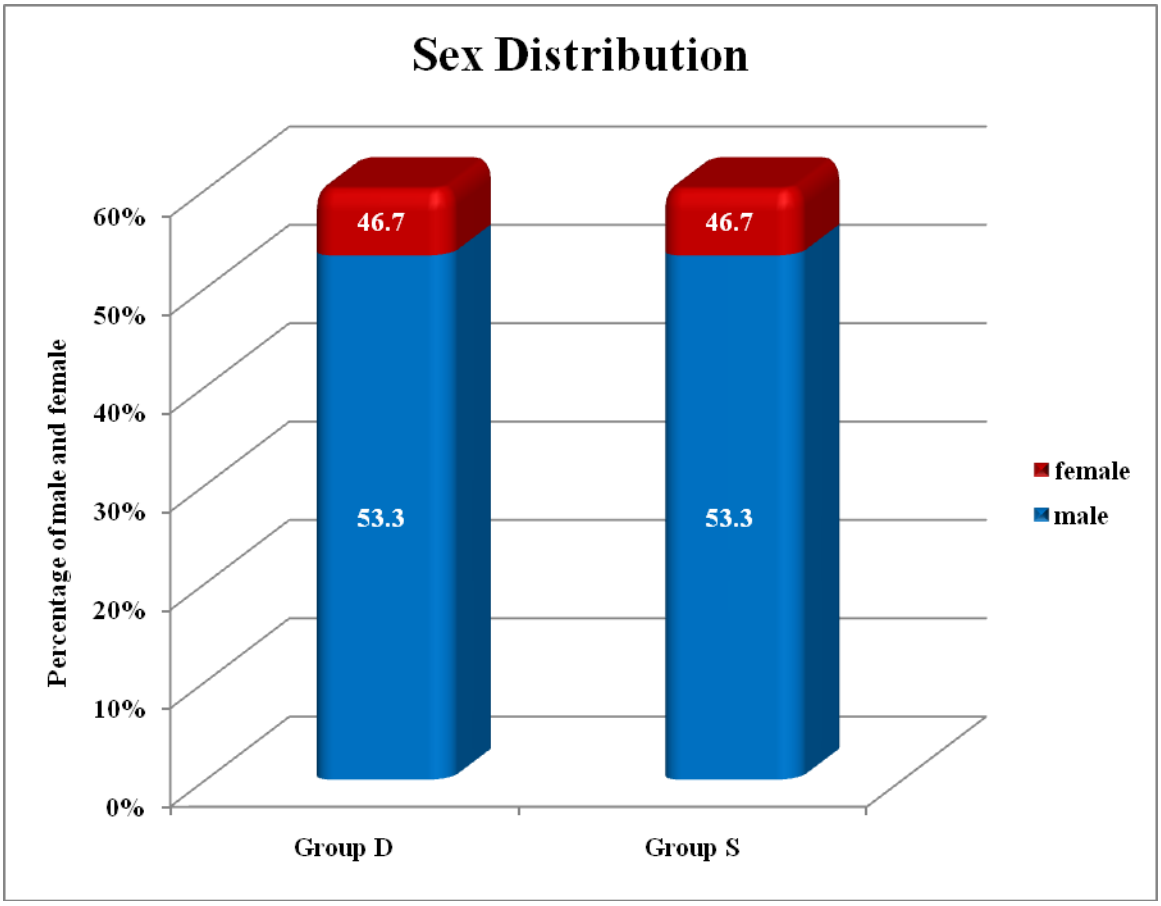

Age distribution
Age in years
40
39.6
39.2
38.8
38.4
38
39.8
39.4
Group D
Group S

Table 2: Sex distribution

Sex	Desflurane group		Sevoflurane group	
	No	**%**	**No**	**%**
Male	16	53.3	16	53.3
Female	14	46.7	14	46.7
Total	30	100	30	100
'p'	1.0 Not significant			

The sex composition of the two groups was identical without any difference.


Sex Distribution
Percentage of male and female
60%
50%
40%
30%
20%
10%
0%
46.7
53.3
46.7
53.3
Group D
Group S
female
male

PROFILE OF CASES STUDIED

Table 3: Diagnosis

Cases	Desflurane group		Sevoflurane group	
	No	**%**	**No**	**%**
Carcinoma breast	**6**	**20**	**4**	**13.3**
Cholelithiasis	**4**	**13.3**	**8**	**26.7**
Epigastric hernia	**3**	**10**	**3**	**10**
Multi nodular goitre Thyroid	**3**	**10**	**4**	**13.3**
Gynaecomastia	**4**	**13.3**	**5**	**16.7**
Solitary nodular goitre Thyroid	**3**	**10**	**1**	**3.3**
Others Bilateral cervical adenitis Bilateral fibroadenoma Bilateral gynaecomastia Bilateral inguinal hernia Fibroadenoma Incisional hernia Pain abdomen for evaluation Right iliac fossa mass Umbilical hernia	**7** - 1 1 - 1 1 1 1 1	**23.3** - 3.3 3.3 - 3.3 3.3 3.3 3.3 3.3	**5** 1 - - 1 1 1 - 1 -	**16.7** 3.3 - - 3.3 3.3 3.3 - 3.3 -
Total	30	100	30	100

Table 4: ASA status

ASA Status	Desflurane group		Sevoflurane group	
	No	**%**	**No**	**%**
1	16	53.3	17	56.7
2	14	46.7	13	43.3
Total	30	100	30	100
'p'	0.7969 Not significant			

There was no significant difference in the ASA status of the two groups. ('p' > 0.05)

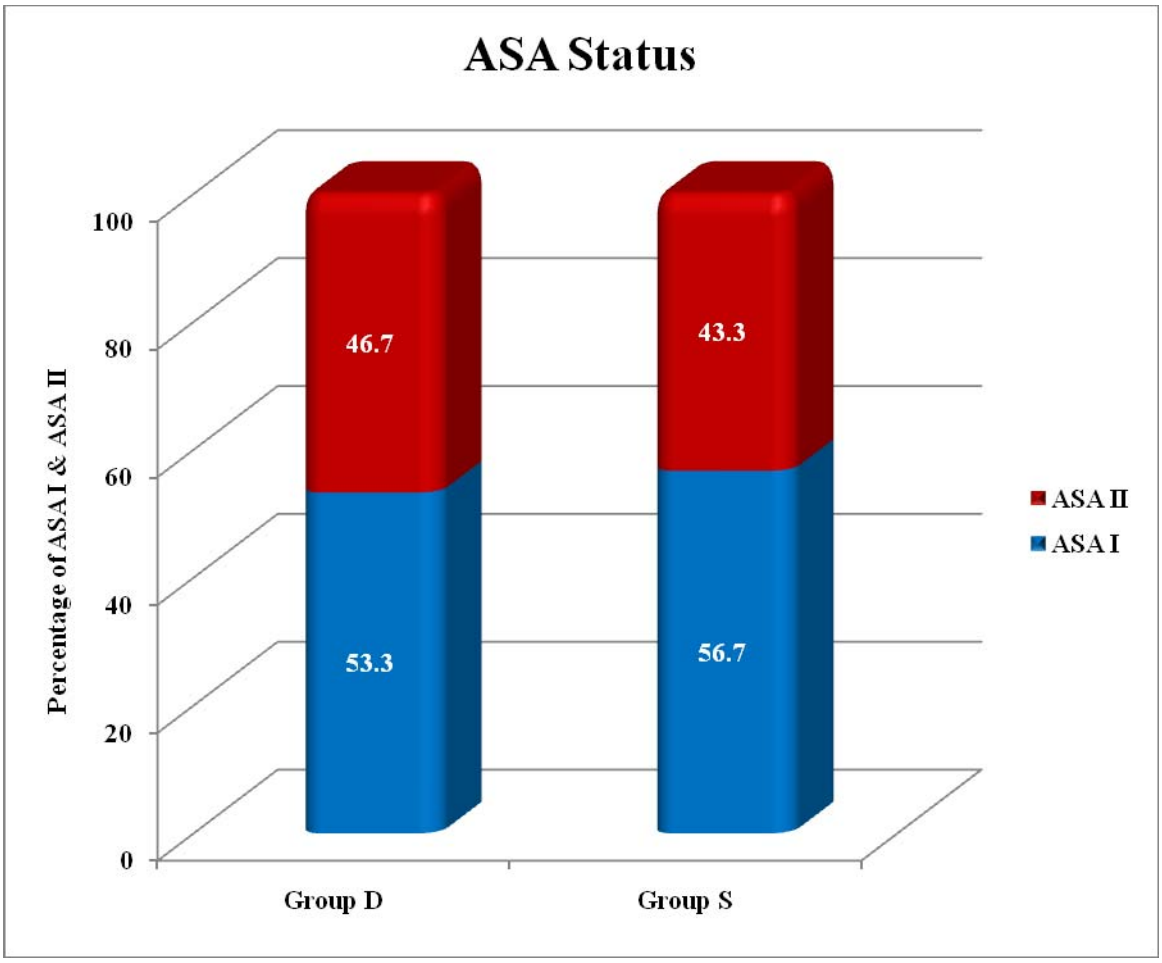

ASA Status
Percentage of ASA I & ASA II
100
80
60
40
20
0
46.7
43.3
53.3
56.7
Group D
Group S
ASA II
ASA I

Table 5: Weight

Parameter	Weight(in kgs)	
	Desflurane Group	**Sevoflurane group**
Range	48 – 58	46 – 58
Mean	52.5	52.2
SD	3.0	3.1
'p'	0.7125 Not Significant	

The weight of the patients was comparable in both the groups.

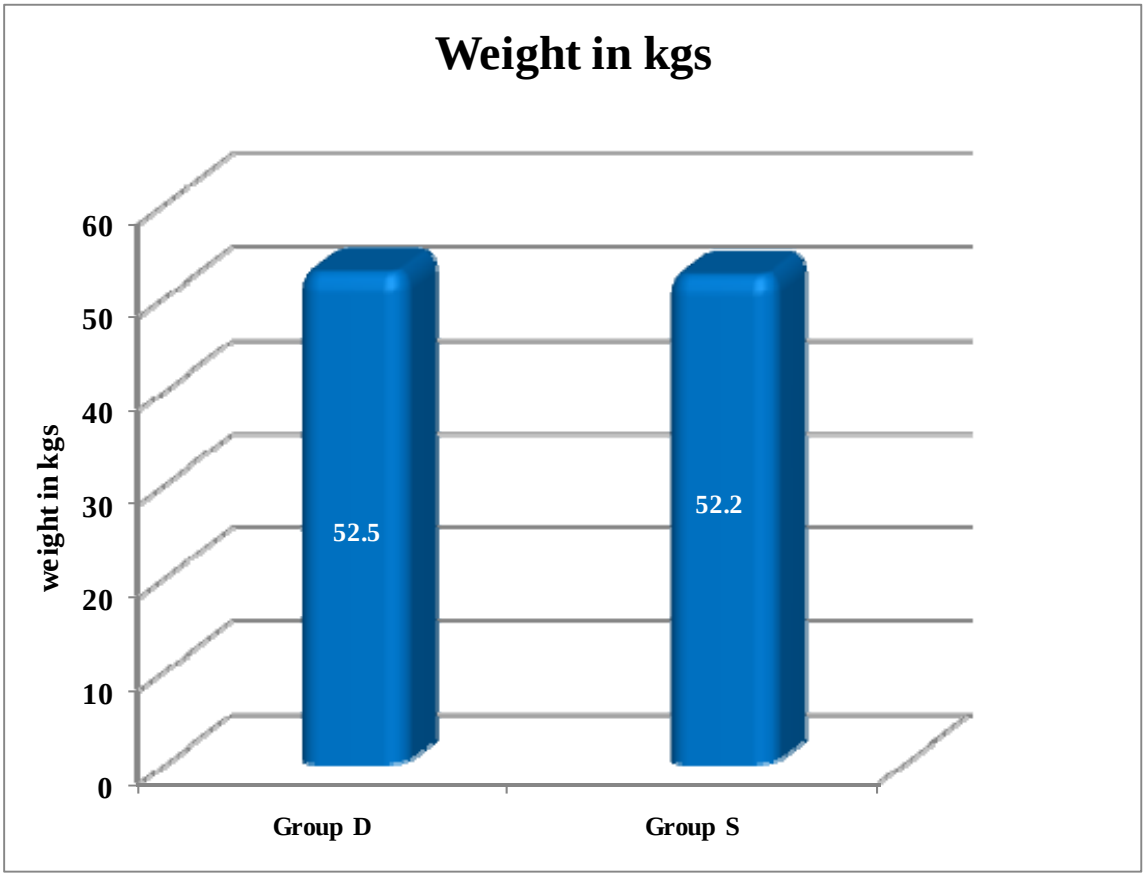

Weight in kgs
weight in kgs
60
50
40
30
20
10
0
52.5
52.2
Group D
Group S

Table 6: Duration of surgery

Parameter	Duration of surgery (in minutes)	
	Desflurane Group	**Sevoflurane group**
Range	80 – 150	80 – 150
Mean	112	110.2
SD	19.4	18.4
'p'	0.5131 Not Significant	

There was no statistically significant difference between the two groups in terms of duration of surgery.

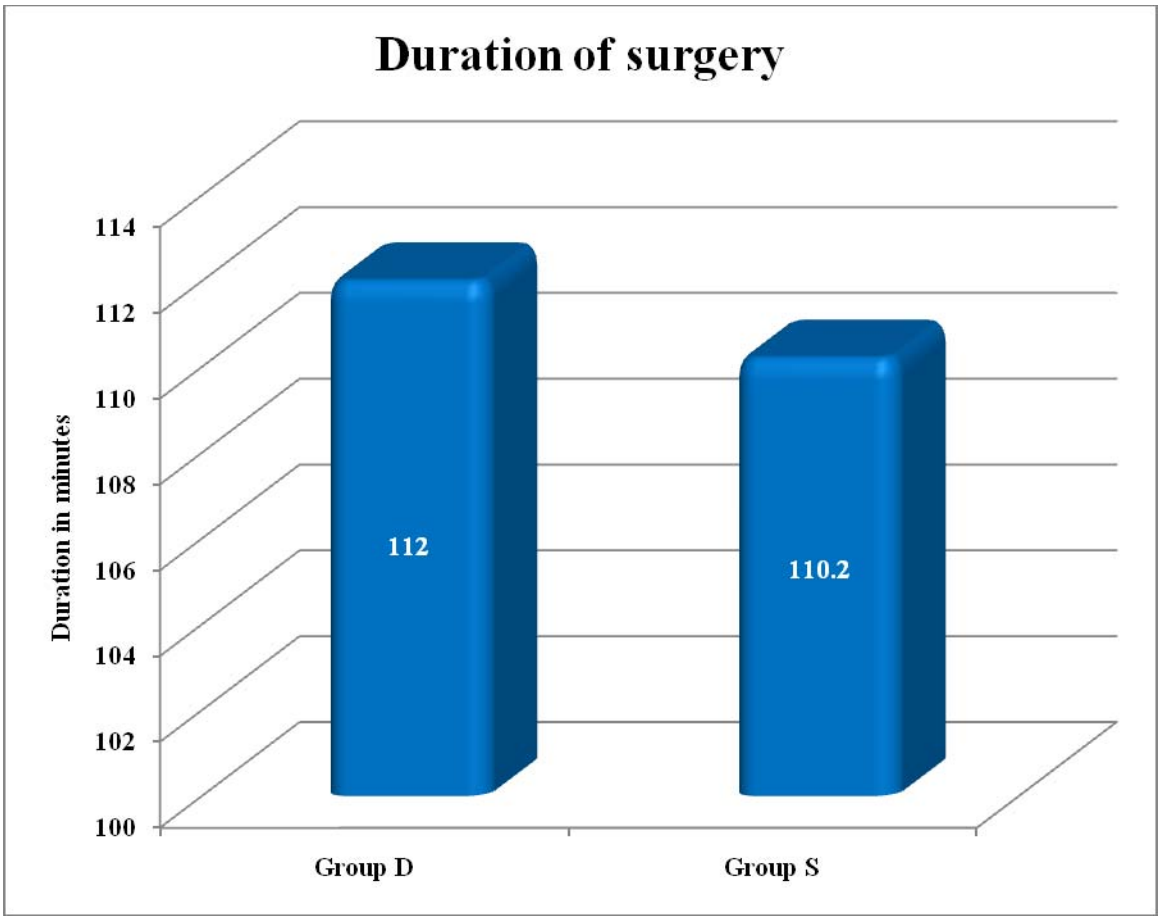
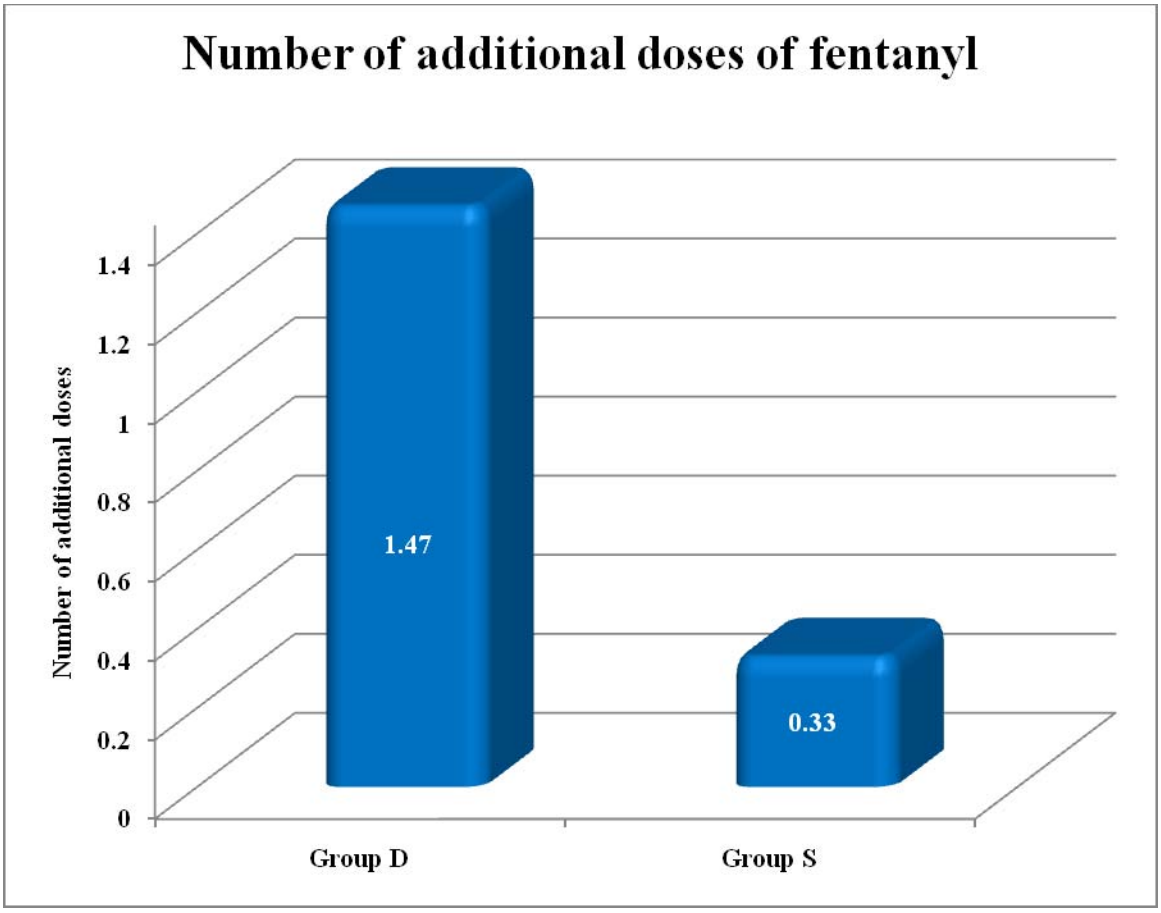

Duration of surgery
Duration in minutes
114
112
110
108
106
104
102
100
112
110.2
Group D
Group S

Table 7: Number of additional doses of fentanyl

Parameter	Number of additional doses of fentanyl	
	Desflurane Group	**Sevoflurane group**
Range	0 – 3	0 – 2
Mean	1.47	0.33
SD	0.9	0.55
'p'	**0.0001 Significant**	

The number of additional doses of fentanyl needed in desflurane group was 1.47 and in sevoflurane group was 0.33. This difference was statistically significant with a p value of 0.0001.


Number of additional doses of fentanyl
Number of additional doses
1.4
1.2
1
0.8
0.6
0.4
0.2
0
1.47
0.33
Group D
Group S

B: Efficacy of the two drugs

Table 8: Time to spontaneous motion

Parameter	Time to spontaneous motion (in minutes)	
	Desflurane Group	**Sevoflurane group**
Range	3 – 5	6 – 9
Mean	4	7.2
SD	0.69	0.76
'p'	**0.0001 Significant**	

Time to spontaneous motion in the desflurane group was 4.0 $\pm$0.69 minutes whereas it was 7.2$\pm$0.76 minutes in the sevoflurane group. This difference was statistically significant (p = 0.0001).

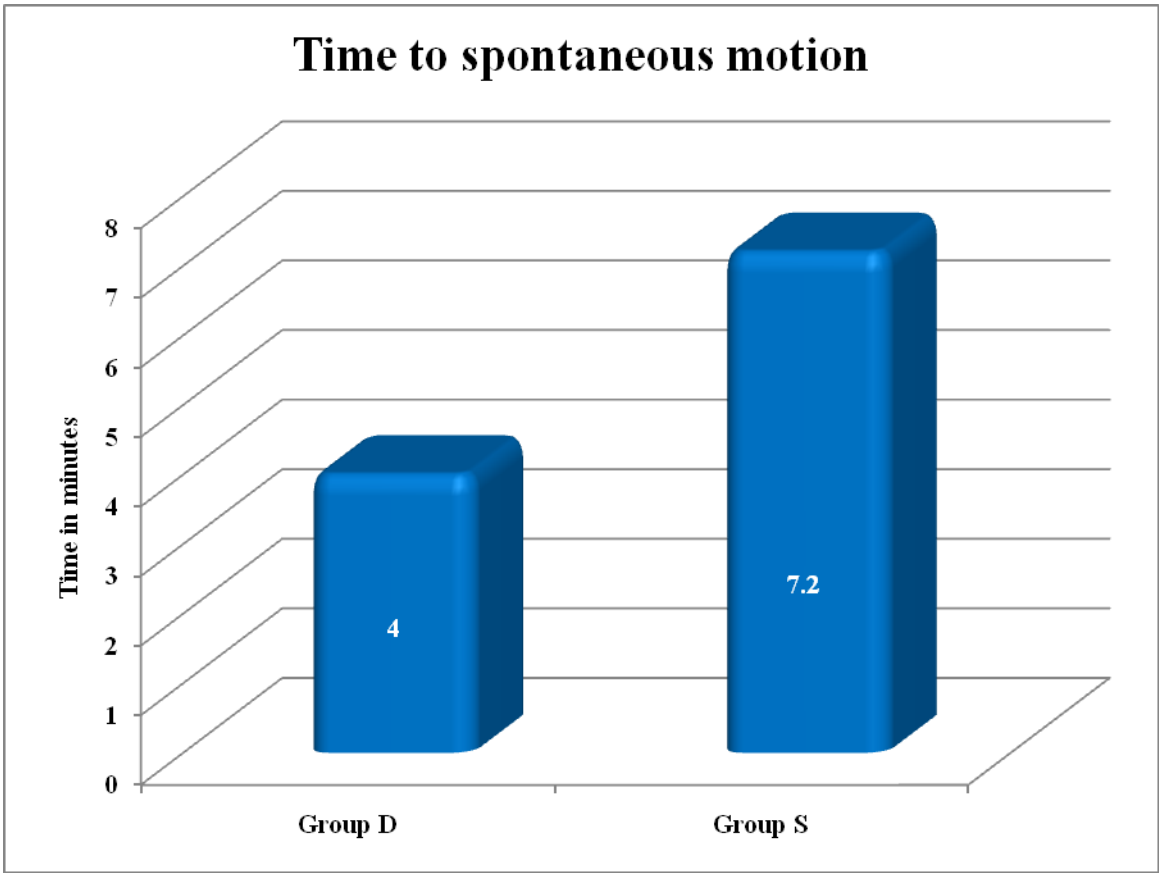

Time to spontaneous motion
Time in minutes
0
1
2
3
4
5
6
7
8
4
7.2
Group D
Group S

Table 9: Time to response to pain

Parameter	Time to response to pain (in minutes)	
	Desflurane Group	**Sevoflurane group**
Range	4 – 7	7 - 11
Mean	5.37	8.57
SD	0.85	0.86
'p'	**0.0001 Significant**	

Time to response to pain in the sevoflurane group (8.57 $\pm$0.86 minutes) was significantly (p = 0.0001) higher than that in the desflurane group (5.37 +0.85 minutes). This difference was statistically significant.

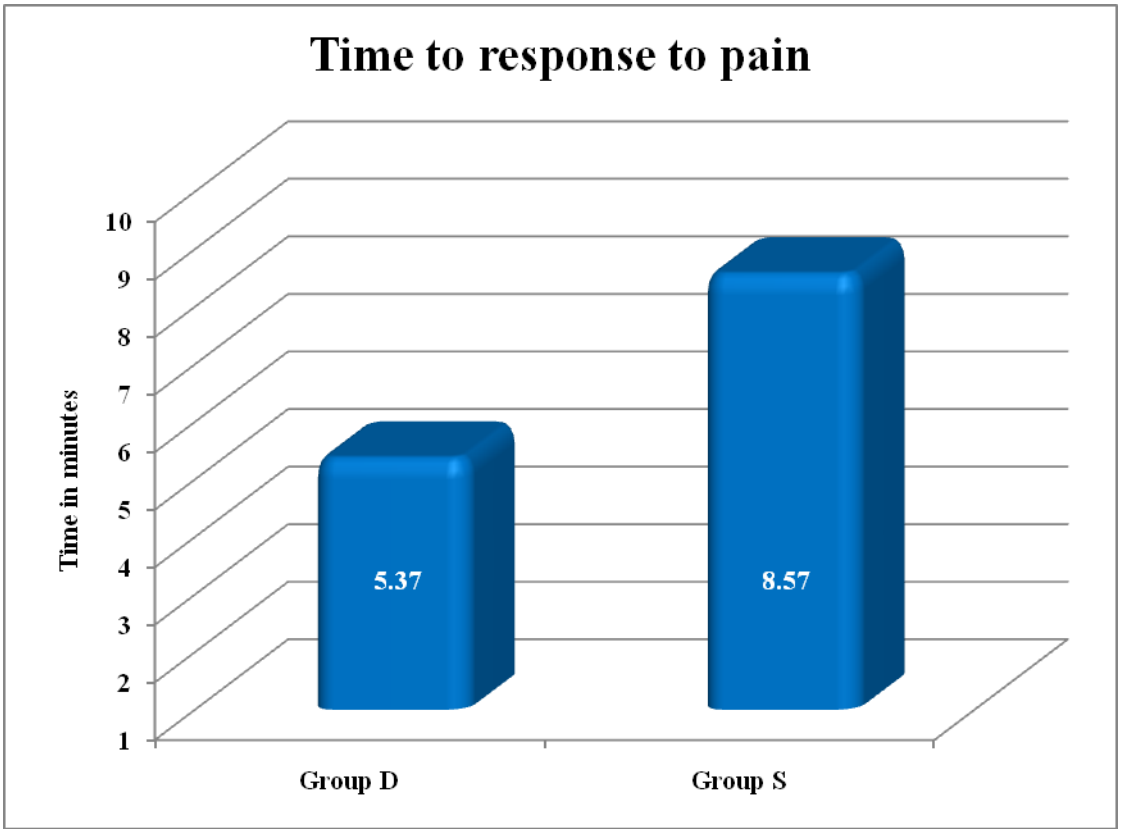

Time to response to pain
Time in minutes
10
9
8
7
6
5
4
3
2
1
5.37
8.57
Group D
Group S

Table 10: Time to extubation

Parameter	Time to extubation (in minutes)	
	Desflurane Group	**Sevoflurane group**
Range	5 – 8	8 - 12
Mean	6.53	10.4
SD	0.82	1.07
'p'	**0.0001 Significant**	

Time to extubation in desflurane group was 6.53minutes when compared to sevoflurane group of 10.4minutes. This difference in the mean time to extubation was statistically significant.

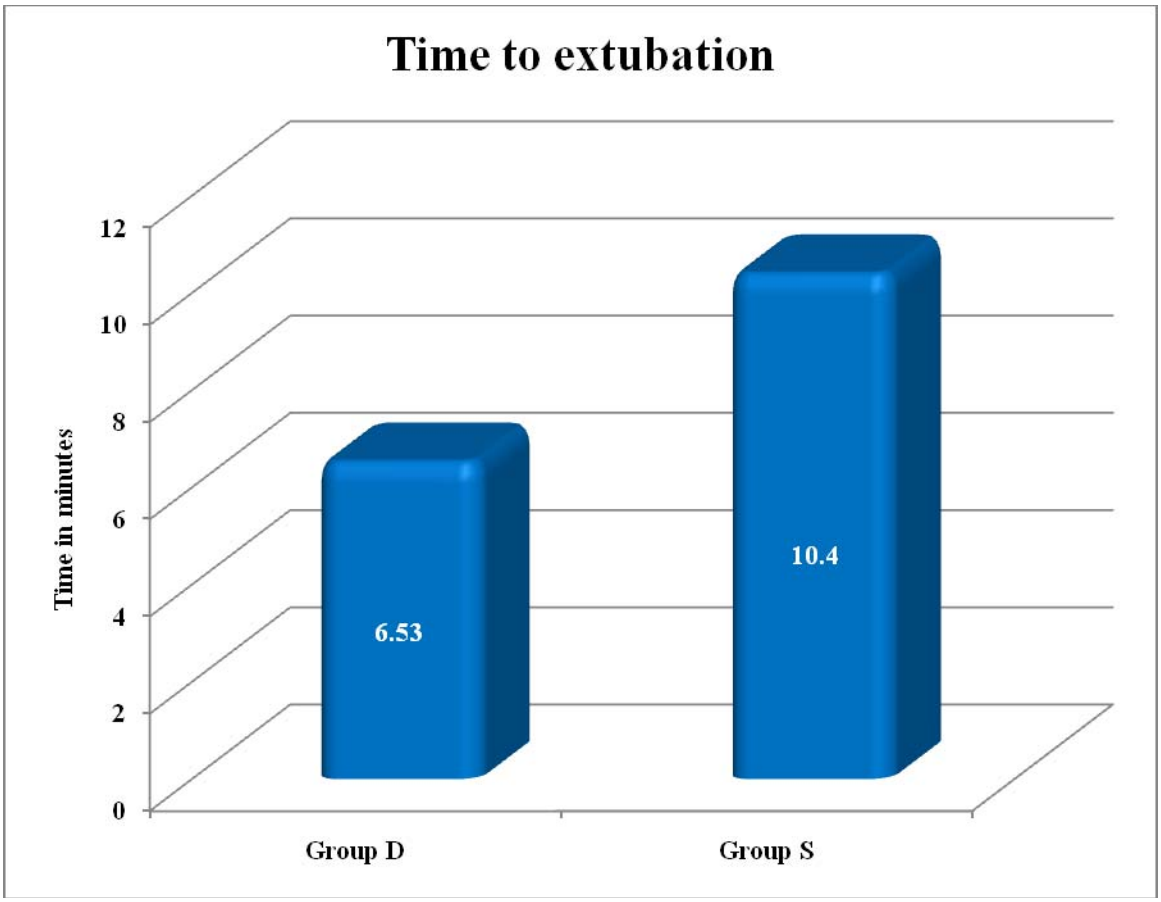

Time to extubation
Time in minutes
0
2
4
6
8
10
12
6.53
10.4
Group D
Group S

Table 11: Time to recall of name

	Time to recall of name (in minutes)	
	Desflurane Group	**Sevoflurane group**
Range	6 – 9	10 – 14
Mean	7.83	12.33
SD	0.79	1.21
'p'	**0.0001 Significant**	

Patients in the desflurane group were able to recall their name in 7.83 $\pm$0.79 minutes. But those in the sevoflurane group were able to recall their name only after 12.33 $\pm$1.21 minutes. This difference was statistically significant ($p = 0.0001$).

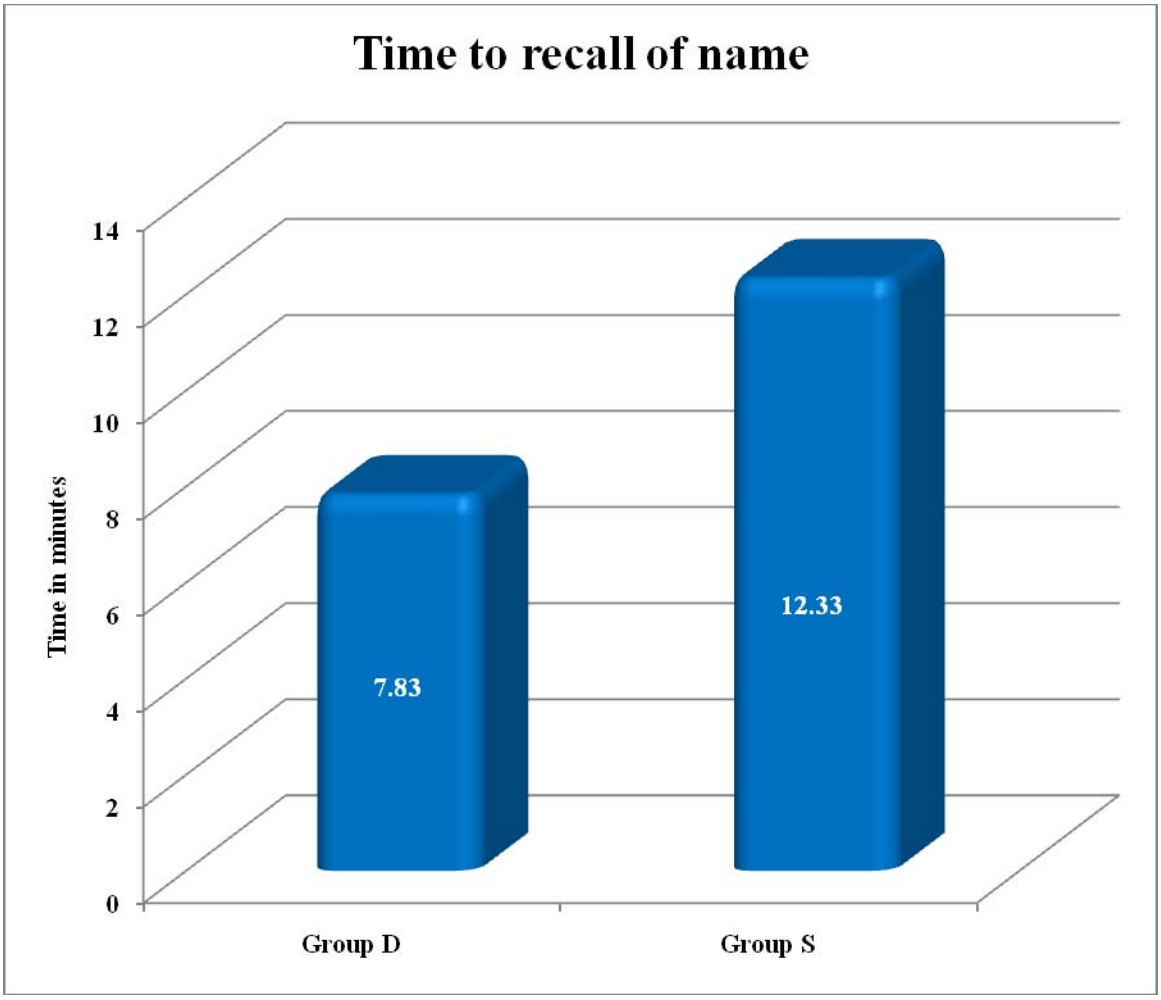

Time to recall of name
Time in minutes
14
12
10
8
6
4
2
0
7.83
12.33
Group D
Group S

Table 12: Time to hand grip

Parameter	Time to hand grip (in minutes)	
	Desflurane Group	**Sevoflurane group**
Range	8 – 11	12 – 17
Mean	9.37	14.27
SD	0.89	1.34
'p'	**0.0001 Significant**	

Time to hand grip in desflurane group was 9.37 $\pm$0.89 minutes and in sevoflurane group was 14.27 $\pm$1.34 minutes. There existed statistically significant difference between the two groups. ('p'=0.0001)

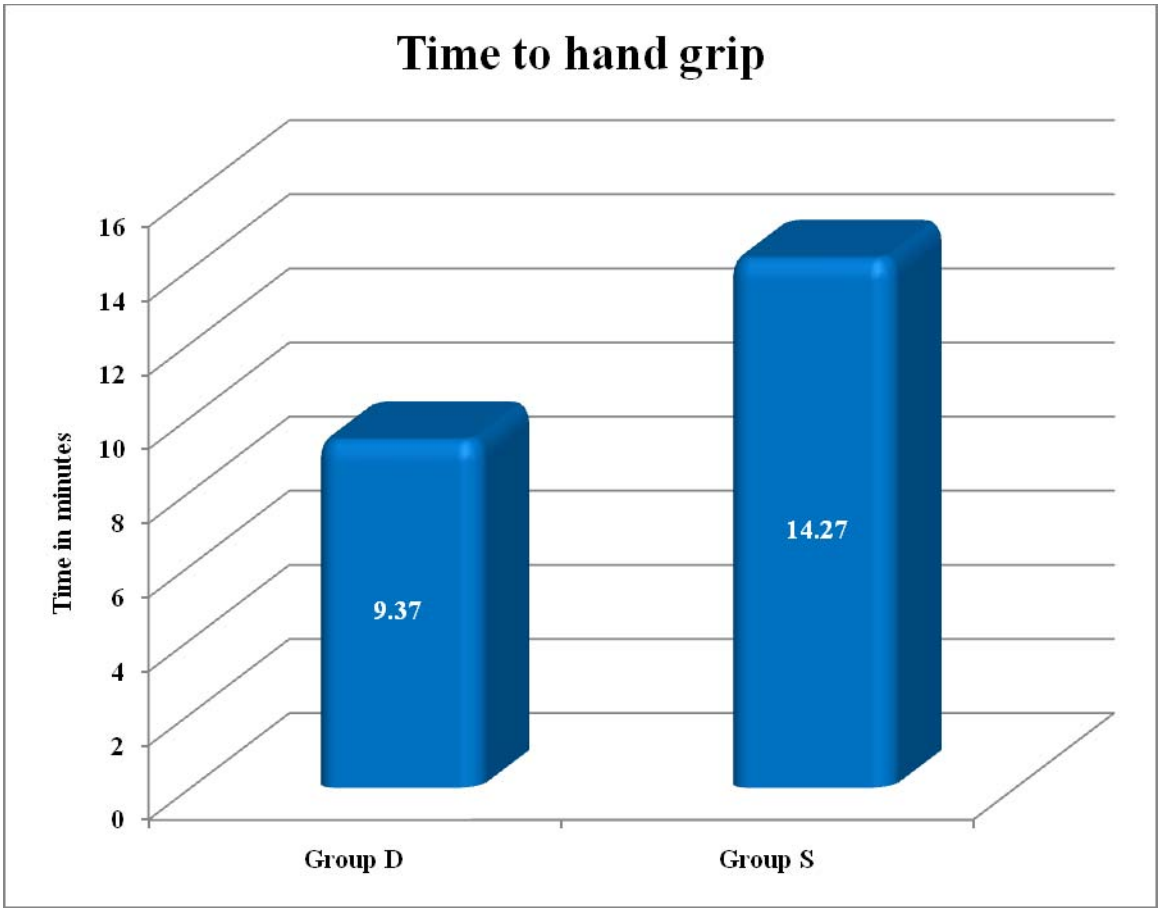

Time to hand grip
Time in minutes
16
14
12
10
8
6
4
2
0
9.37
14.27
Group D
Group S

Table 13: Time to achieve PARS > 10

Parameter	Time to achieve PARS > 10 (in minutes)	
	Desflurane Group	**Sevoflurane group**
Range	9 – 12	14 - 19
Mean	10.47	16.63
SD	0.86	1.47
'p'	**0.0001 Significant**	

Patients in the desflurane group achieved PARS > 10 at 10.47 $\pm$0.86 minutes whereas the patients in the sevoflurane group were able to achieve it only after 16.63 $\pm$1.47 minutes. This difference was statistically significant ($p < 0.05$).

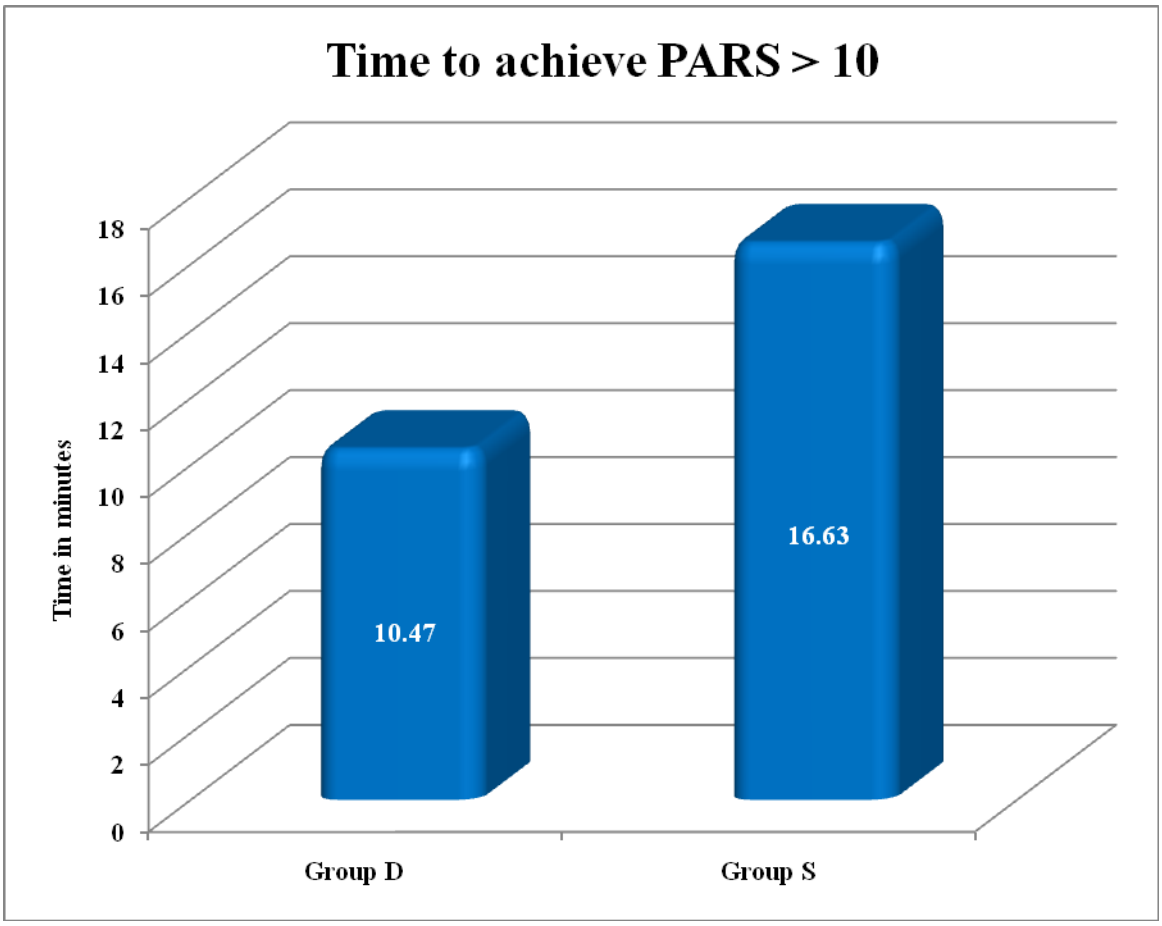

Time to achieve PARS > 10
Time in minutes
0
2
4
6
8
10
12
14
16
18
10.47
16.63
Group D
Group S

Table 16: Changes in Pulse Rate

Variable			'p'
	Desflurane Group	Sevoflurane group	
Baseline Pulse Rate	82.7 ± 7.7	84.2 ± 8.2	0.4092 Not significant
Intra operative Pulse Rate	82.7 ± 7.2	80.1 ± 8.1	0.2704 Not significant

Baseline pulse rate and intraoperative pulse rate were comparable between the desflurane and sevoflurane groups. There was no statistically significant difference.

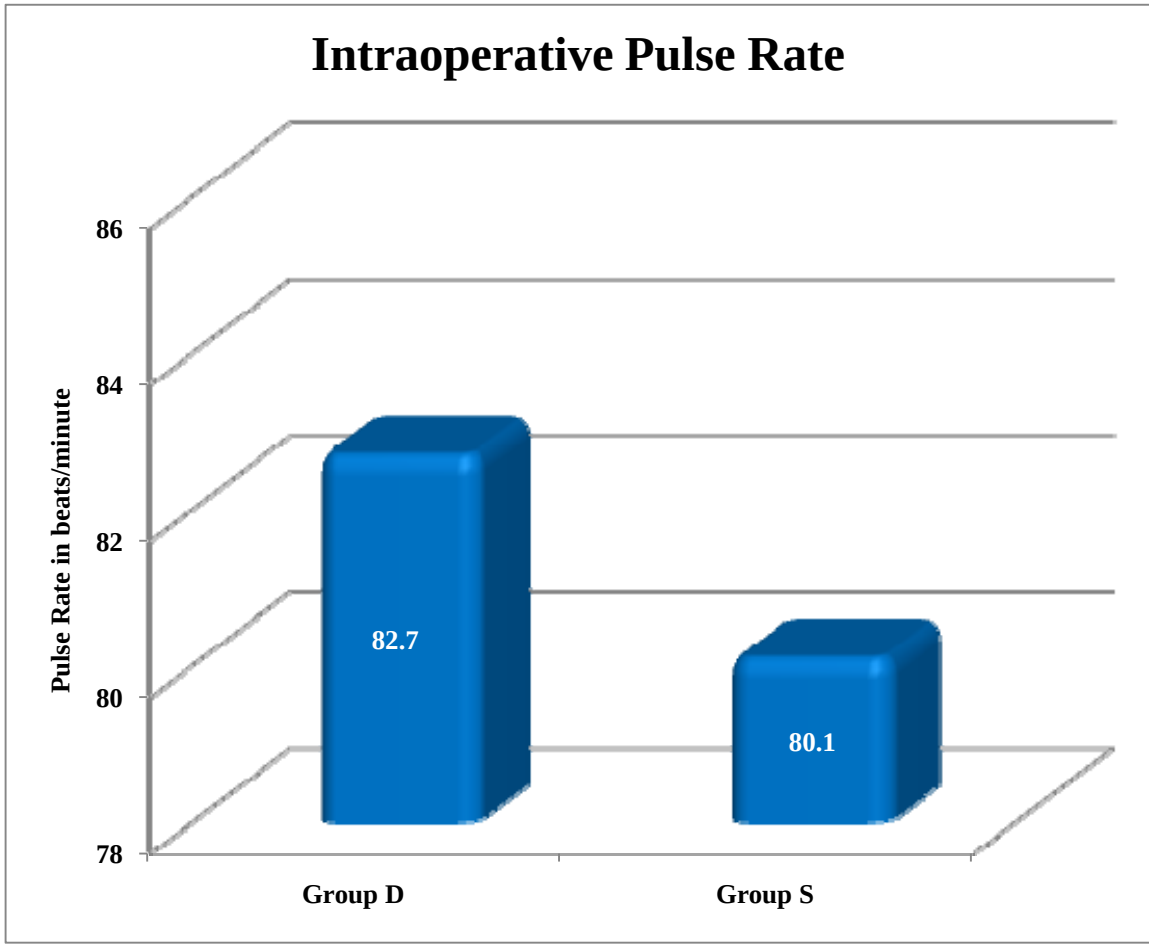
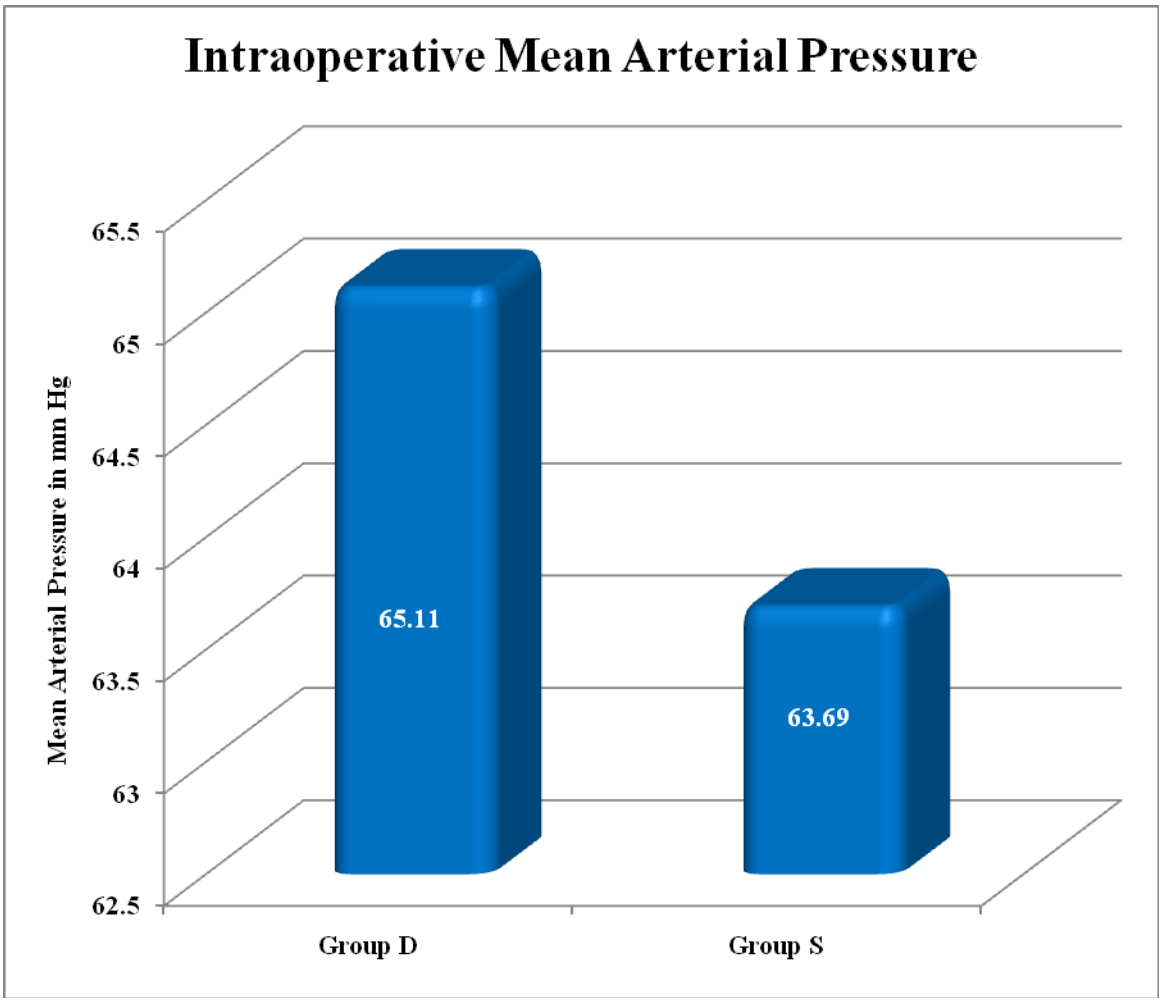

Intraoperative Pulse Rate
Pulse Rate in beats/minute
86
84
82
80
78
82.7
80.1
Group D
Group S

Table 17: Changes in Mean Arterial Pressure

Variable	Changes in Mean Arterial Pressure mmHg		'p'
	Desflurane Group	**Sevoflurane group**	
Baseline Mean Arterial Pressure	64.7 ± 3.2	65.3 ± 3	0.4097 Not significant
Intra operative Mean Arterial Pressure	65.11 ± 3.27	63.69± 2.96	0.06849 Not significant

The baseline and intraoperative mean arterial pressure in both desflurane and sevoflurane groups were comparable with no statistically significant difference.


Intraoperative Mean Arterial Pressure
Mean Arterial Pressure in mm Hg
65.5
65
64.5
64
63.5
63
62.5
65.11
63.69
Group D
Group S

DISCUSSION

General anesthesia is popular among the surgeons, anaesthesiologists, and patients and still remains the mainstay of anesthesia in many centres. With the introduction of less soluble volatile anesthetics which promote early recovery and also maintains hemodynamics and provide amnesia makes general anesthesia the technique of choice for many patients.

It is desirable to have a faster recovery from anesthesia. This study compared the hemodynamic, emergence and recovery characteristics of sevoflurane with desflurane in general anesthesia.

The time to spontaneous motion, eye opening, response to pain were shorter in the desflurane group. The time to extubation, recall of name, and hand grip were also shorter in the desflurane group compared to sevoflurane group. Post anesthesia recovery score of greater than 10 was achieved earlier in the desflurane group.

In the desflurane group, patient moved their limbs in a mean time of 4minutes after the discontinuation of the anesthetics whereas it took a mean of 7.2minutes in the sevoflurane group.

The time to response to pain was achieved in a mean time of 5.37minutes in the desflurane group whereas in the sevoflurane group it took a mean of 8.57minutes.

The patients in the desflurane group were extubated earlier than those in the sevoflurane group. The patients in the desflurane group were able to recall their names in a mean time of 7.83minutes whereas those in the sevoflurane group took 12.33minutes.

The time to hand grip was achieved earlier in the desflurane group. The post anesthesia recovery score of greater than 10 (PARS>10) was achieved in a mean of 10.47minutes in desflurane group which was earlier than sevoflurane.

The study by **Nathanson et al.** suggested that sevoflurane and desflurane provided similar intraoperative conditions during the maintenance period. Although early recovery was more rapid after desflurane, there was no difference in later recovery end-points.

Randomised, double-blind study of **Tarazi et al.** showed that both sevoflurane and desflurane were acceptable inhalational anaesthetics for outpatient tubal ligation surgery.

In this study there was no significant difference in the recovery times between the two groups after 30minutes.

Song et al. found that the late recovery profiles and incidences of postoperative side effects were similar after desflurane and sevoflurane. It was also showed that regardless of the duration of anaesthesia, elimination was faster and recovery was quicker for the inhaled anaesthetic desflurane than for the inhaled anaesthetic sevoflurane.

Hemodynamic profile

Both the desflurane and sevoflurane maintained the hemodynamics within 20% of the baseline values, but desflurane required more number of additional doses of fentanyl than sevoflurane.

In the desflurane group, hemodynamics could not be maintained with the additional doses of fentanyl in 3 patients and they were excluded from the study, were as in the sevoflurane group, only 1 patient was excluded from the study.

Hypotension was easily managed with fluids and blood replacement and none of the patients were excluded in both the groups.

SUMMARY

The aim of this study is to prospectively compare the hemodynamic, emergence and recovery characteristics of sevoflurane with that of desflurane in general anesthesia. 60 ASA I and II patients undergoing elective surgical procedures less than 3 hours duration under endotracheal general anesthesia were randomly divided into two groups. Both the groups were induced with standard intravenous induction technique. Group D was maintained with 3% desflurane and group S with 1% sevoflurane in 50% oxygen with 50% nitrous oxide. Injection fentanyl 0.5µg/kg was repeated every 30 minutes. Injection vecuronium 0.02mg/kg was repeated every 30 minutes. Mean arterial pressure and heart rate were noted before induction and every 5 minutes after induction. If there is any increase in the mean arterial pressure and heart rate more than 20% of the preinduction values, an additional dose of injection fentanyl 1µg/kg was given. If there is any reduction in the mean arterial pressure more than 20% from the baseline value, it was treated with bolus of intravenous fluids and replacement of intraoperative blood loss. When the hemodynamics of the patient was unresponsive to the above measures, the patient was excluded from the study. Nitrous oxide and volatile anesthetic were discontinued after the last skin suture. Residual neuromuscular

blockade was reversed with injection neostigmine 40µg/kg and injection glycopyrrolate 10µg/kg intravenously. The following were noted and recorded.

The time of discontinuation of anesthetic agents were noted as time zero for all the subsequent measurements and recovery times were determined at 1-minute intervals to awakening.

Number of additional doses of fentanyl needed was noted.

Time to – 1. First spontaneous motion

2. Response to painful pinch

3. Extubation

4. Recall of name

5. Hand grip

6. Achieve a PARS> 10 (post anesthesia recovery score of Aldrete and Kroulik) were noted.

The following results were obtained. Of the two groups compared,

1. Age, sex, weight and the duration of surgery were comparable in both the groups.
2. Both desflurane and sevoflurane maintained hemodynamics, but desflurane needed more number of additional doses of fentanyl to

maintain the hemodynamic stability. This difference was found to be **statistically significant.**

3. The time to first spontaneous motion, response to pain, extubation, recall of name, and hand grip were shorter in the desflurane group than the sevoflurane group. The difference was **statistically significant**.
4. The time to achieve a PARS of greater than 10 was earlier in the desflurane group and it was **statistically significant**.

CONCLUSION

In conclusion, desflurane provides earlier emergence and recovery from anesthesia compared to sevoflurane. Both desflurane and sevoflurane maintained hemodynamic stability, but to maintain the hemodynamics desflurane needed more number of additional doses of fentanyl.

BIBLIOGRAPHY

1. Eger II EI, Gong D, Koblin DD, et al: Effect of anesthetic duration on kinetic and recovery characteristics of desflurane vs. sevoflurane (plus compound A) in volunteers. *Anesth Analg* 1998; 86:414-421.
2. McKay RE, Large MJC, Balea MC, McKay WR: Airway reflexes return more rapidly after desflurane anesthesia than after sevoflurane anesthesia. *Anesth Analg* 2005; 100:697-700.
3. Neumann MA, Weiskopf RB, Gong DH, et al: Changing from isoflurane to desflurane towards the end of anesthesia does not accelerate recovery in humans. *Anesthesiology* 1998; 88:914-921.
4. Nordmann GR, Read JA, Sale SM, et al: Emergence and recovery in children after desflurane and isoflurane anaesthesia: Effect of anaesthetic duration. *Br J Anaesth* 2006; 96:779-785.
5. Eger EI II, Bowland T, Ionescu P, et al. Recovery and kinetic characteristics of desflurane and sevoflurane in volunteers after 8-h exposure, including kinetics of degradation products. *Anesthesiology* 1997; **87**: 517–26.
6. Juvin P, Vadam C, Malek L, et al. Postoperative recovery after desflurane, propofol, or isoflurane anesthesia among morbidly obese patients: a prospective, randomized study. *Anesth Analg* 2000; **91**:714.

7. Song D, Joshi GP, White PF. Fast-track eligibility after ambulatory anesthesia: a comparison of desflurane, sevoflurane, and propofol. *Anesth Analg* 1998; **86**: 267–73.
8. Beaussier M, Deriaz H, Abdelahim Z, et al. Comparative effects of desflurane and isoflurane on recovery after long lasting anaesthesia. *Can J Anaesth* 1998; **45**: 429–34.
9. Nathanson MH, Fredman B, Smith I, White PF. Sevoflurane versus desflurane for outpatient anesthesia: a comparison of maintenance and recovery profiles. *Anesth Analg* 1995; **81**: 1186–90.
10. Smiley R, Ornstein E, Mateo RS, et al. Desflurane and isoflurane in surgical patients: comparison of emergence time. *Anesthesiology* 1991; **74**: 425–8.
11. Chen X, Zhao M, White PF, et al. The recovery of cognitive function after general anesthesia in elderly patients: a comparison of desflurane and sevoflurane. *Anesth Analg* 2001; **93**: 1489–94.
12. Tarazi EM, Philip BK. A comparison of recovery after sevoflurane or desflurane in ambulatory anesthesia. *J Clin Anesth* 1998; **10**: 272–7.
13. Faster wash-out and recovery for desflurane vs. sevoflurane in morbidly obese patients when no premedication is used Br J Anaesth September 1, 2007 99:353-358.

14. Emergence and Recovery Characteristics of Desflurane Versus Sevoflurane in Morbidly Obese Adult Surgical Patients: A Prospective, Randomized Study Anesth. Analg. December 1, 2004 99: 1848-1853.
15. Ghouri AF, Matthew B, White PF. Recovery profiles after desflurane-nitrous oxide versus isoflurane-nitrous oxide in outpatients. *Anesthesiology* 1991;**74**: 419–24.
16. Graham SG, Aitkenhead AR. A comparison between propofol and desflurane anaesthesia for minor gynaecological laparoscopic surgery. *Anaesthesia* 1993; **48**: 471–5.
17. Dexter F, Tinker JH. Comparison between desflurane and isoflurane or propofol on time to following commands and time to discharge. A metaanalysis. Anesthesiology 1995;83:77 – 82.
18. Tang J, White PF, Wender RH, et al. Fast-track office-based anesthesia: a comparison of propofol versus desflurane with antiemetic prophylaxis in spontaneously breathing patients. Anesth Analg 2001;92:95–9.
19. Meta-analysis of trials comparing postoperative recovery after anesthesia with sevoflurane or desflurane Am J Health Syst Pharm January 1,2005 62:63-68.

20. Miller RD : Miller's anesthesia 7th ed. New York, Churchill Livingstone 2009 pp 515-530.
21. Clinical anaesthesia Paul.G. Barash, Bruce.F.Cullen, Robert.K.stoelting 5th ed. Pp 414 - 440
22. Earl M. Strum, Janos Szenohradszki, MD, PhD, et al. Emergence and Recovery Characteristics of Desflurane Versus Sevoflurane in Morbidly Obese Adult Surgical Patients: A Prospective, Randomized Study. Anesth&Analg December 2004 vol.99 no.6 1848-1853.
23. Smith I, Taylor E, White PF. Comparison of tracheal extubation in patients deeply anesthetized with desflurane or isoflurane. *Anesth Analg* 1994; **79**:642–5
24. Naidu-Sjösvärd K, Sjöberg F, Gupta A. Anesthesia for video arthroscopy of the knee: a comparison between desflurane and sevoflurane. *Acta Anaesthesiol Scand* 1998; **42**: 464–71.
25. Grundman U, Silomon M, Bach F, Becker S, Bauer M, Larsen, Kleinschmidt S. Recovery profile and side effects of remifentanil-based anaesthesia with desflurane or propofol for laparoscopic cholecystectomy. Acta Anaesthesiol Scand 2001; 45: 320-326.

26. Bennett JA, Mahadeviah A, Stewart J, Lingaraju N, Keykhah MM: Desflurane controls the hemodynamic response to surgical stimulation more rapidly than isoflurane. J Clin Anesth 1995; 7: 288-291.

27. Loop T, Priebe HJ. Recovery after anesthesia with remifentanil combined with propofol, desflurane, or sevoflurane for otorhinolaryngeal surgery. Anesth Analg 2000; Jul; 91 (1): 123-129.

28. Larsen B, Seitz A, Larsen R. Recovery of cognitive function after remifentanil-propofol anesthesia: a comparison with desflurane and sevoflurane anesthesia. Anesth Analg 2000; Jan; 90 (1): 168-174.

29. Dupont J, Tavernier B, Ghosez Y, Durinck L, Thevenot A, Moktadir-Chalons N, Ruyffelaere- Moises L, Declerck N, Scherpereel P. Recovery after anaesthesia for pulmonary surgery: desflurane, sevoflurane and isoflurane. Br J Anaesth 1999; 82: 355-359.

30. A Practice of Anesthesia Wylie and Churchill-Davidson's, 7TH Ed pp 523 – 536.

PROFORMA

COMPARISON OF HEMODYNAMIC, EMERGENCE AND RECOVERY CHARACTERISTICS OF SEVOFLURANE WITH DESFLURANE

NAME: AGE: IPNO:

DIAGNOSIS: PROCEDURE:

ASA: I / II WEIGHT:

DURATION OF THE SURGERY:

PREMEDICATION: Inj.midazolam 0.05mg/kg IV

Inj.glycopyrollate 10µg/kg IV

PREOPERATIVE: Mean arterial blood pressure:

Pulse rate:

GENERAL ANESTHESIA: Induction: Inj.thiopentone 5mg/kg

Inj.fentanyl 2µg/kg

Inj.succinylcholine 1.5mg/kg

Injection fentanyl 0.5µg/kg repeated every 30mins after induction.

Neuromuscular blockade maintained with injection vecuronium 0.1mg/kg bolus dose followed by 0.02mg/kg every 30minutes.

GROUP: DESFLURANE/SEVOFLURANE

DESFLURANE GROUP: 3% desflurane in 50% oxygen and 50% nitrous oxide.

SEVOFLURANE GROUP: 1% sevoflurane in 50% oxygen and 50% nitrous oxide.

If there is any increase in the mean arterial pressure and heart rate more than 20% of the preinduction values, a rescue dose of injection fentanyl 1µg/kg was given.

Time	Mean arterial pressure	Heart rate
Pre-induction		
5minutes		
10minutes		
15minutes		
20minutes		
25minutes		
30minutes		
35minutes		
40minutes		
45minutes		
50minutes		
55minutes		
60minutes		

Parameters observed:

Number of rescue doses of fentanyl needed:

Parameter- Time to	Time in minutes
First spontaneous motion	
Response to painful pinch	
Extubation	
Recall of name	
Hand grip	
Achieve a PARS > 10.	

GROUP - D (Desflurane Group)

Sl. No.	Name	age	sex	weight	ip no	diagnosis	asa status	blood sugar	blood urea	serum creatinine	serum sodium	serum potassium
1	mani	19	m	56	6405	cholelithiasis	1	72	24	0.9	134	3.5
2	muthu	28	f	52	7710	sng thyroid	1	78	20	0.8	136	3.3
3	raja	50	m	50	2270	epigastric hernia	1	76	28	0.7	142	4.1
4	murugeswari	37	f	54	5587	ca breast	2	88	26	1	137	3.6
5	chitra	28	f	48	5917	b/l fibroadenoma	2	102	30	0.9	143	3.7
6	kaliammal	57	f	51	5932	incisional hernia	1	112	30	0.8	138	3.8
7	ayyanar	41	m	56	8946	gynaecomastia	1	96	28	1.2	134	3.6
8	bose	54	m	48	5629	cholelithiasis	2	92	22	0.8	135	3.5
9	poongodi	24	f	48	5876	sng thyroid	1	100	24	0.9	136	3.6
10	bagyam	45	f	50	6453	mng thyroid	1	86	28	0.9	135	3.3
11	thangam	38	f	52	6618	ca breast	2	108	22	0.8	133	3.8
12	rajan	47	m	56	7153	rif mass	2	104	20	0.9	136	4
13	amirtha	50	f	52	6827	ca breast	2	68	22	0.9	135	4.2
14	banu	52	f	50	5613	ca breast	2	72	24	0.7	136	3.7
15	kannan	34	m	56	7324	gynaecomastia	1	78	32	0.9	137	3.8
16	muthu	56	m	50	5647	epigastric hernia	2	88	28	0.8	142	3.5
17	murugan	32	m	58	6702	mng thyroid	1	118	34	1.2	141	3.6
18	palani	45	m	52	6134	cholelithiasis	1	112	30	0.9	142	3.7
19	parimala	38	f	48	6713	sng thyroid	1	108	22	0.8	136	3.6
20	petchi	25	f	50	7215	fibroadenoma	1	110	26	0.9	137	3.8
21	mari	45	f	52	6317	ca breast	2	98	22	0.8	136	3.7
22	surya	23	m	54	5438	gynaecomastia	2	94	32	1.2	135	3.6
23	meena	40	f	50	4876	mng thyroid	1	98	34	1	133	3.5
24	subash	32	m	54	6721	cholelithiasis	2	76	22	0.8	134	3.3
25	maruthu	53	m	56	5317	pain abdomen	2	78	24	0.9	137	3.5
26	mani	36	m	58	4637	b/l gynaecomastia	2	86	22	0.8	135	3.7
27	ashok	27	m	54	5134	gynaecomastia	1	88	24	0.7	138	3.8
28	rani	43	f	52	6120	ca breast	2	78	22	0.8	136	3.6
29	kulandaivelu	50	m	54	6112	umbilical hernia	1	95	24	0.9	135	3.5
30	muthukumar	32	m	54	6218	epigastric hernia	1	92	20	0.7	134	3.5

GROUP - D (Desflurane Group)

baseline PR	baseline MAP	intraop PR	intraop MAP	No.of Additional doses of Fentanyl	duration of Surgery	time to spontaneous motion	time to response to pain	time to extubation	time to recall of name	time to hand grip	time to achieve PARS>10
76	66	74.6	63.3	1	120mins	4	5	8	9	11	11
72	64	72.8	66	2	95mins	3	5	6	7	9	10
78	58	76.6	58	nil	100mins	4	4	7	8	10	12
82	66	80.8	64.2	1	90mins	4	6	7	8	11	11
84	62	82.2	64.8	2	120mins	5	7	8	9	11	12
86	68	84.4	66	1	140mins	5	5	6	8	9	10
88	70	86.2	70.2	nil	100mins	5	7	7	8	9	11
72	68	72.6	70.8	3	120mins	3	4	5	7	8	9
76	66	76.4	66.6	1	100mins	4	5	8	9	11	11
74	64	74.2	64.8	1	95mins	4	4	5	6	8	9
92	62	80.8	60.2	nil	100mins	4	6	7	8	9	10
90	58	88.6	60.4	1	150mins	5	7	8	9	10	11
94	66	90.8	64.6	nil	120mins	4	5	6	8	9	11
96	60	92.8	60.2	1	120mins	4	6	7	8	9	11
88	64	86.8	65.3	1	100mins	4	5	6	8	9	11
72	62	76.4	60	2	120mins	5	6	7	9	10	12
76	66	74.6	64.6	1	110mins	3	5	6	8	9	10
72	62	76.4	64	3	110mins	4	5	7	8	10	11
76	62	78.2	64.3	3	120mins	4	6	6	7	9	10
74	64	70.8	64.6	2	140mins	5	6	7	9	10	11
82	66	82.8	66.8	2	150mins	3	4	6	7	8	9
84	68	84.2	68.2	1	110mins	4	5	6	8	9	10
84	70	88.6	68	3	90mins	4	6	7	8	10	11
88	70	88.8	72.4	2	110mins	4	5	6	7	9	10
92	66	94.6	66.4	2	120mins	3	5	6	8	9	10
80	62	82.4	64.3	2	120mins	3	5	6	7	8	9
92	68	92.2	68.2	1	110mins	5	6	7	8	10	11
96	66	96.2	68	1	110mins	4	6	6	7	9	10
84	64	88.6	62	2	90mins	3	5	6	7	9	10
82	64	84.4	66.2	2	80mins	4	5	6	7	9	10

GROUP - S (SEVOFLURANE GROUP)

Sl. No.	Name	age	sex	weight	ip no	diagnosis	asa status	blood sugar	blood urea	serum creatinine	serum sodium	serum potassium
1	pandi	20	m	54	5481	gynaecomastia	2	76	22	0.8	135	3.6
2	paulraj	32	m	52	6233	epigatric hernia	2	80	28	0.8	134	3.5
3	arumugam	49	m	56	5601	cholelithiasis	1	88	24	0.9	133	3.5
4	mari	38	f	50	6108	mng thyroid	1	86	26	0.7	136	4.2
5	muthu	30	f	48	7324	sng thyroid	1	96	30	1	137	3.9
6	karupayee	60	f	46	7642	incisional hernia	2	98	34	0.9	138	3.3
7	subbiah	40	m	50	6435	cholelithiasis	1	92	26	0.9	142	3.5
8	paneer	52	m	50	8120	b/l inguinal hernia	2	88	28	0.9	144	3.6
9	asairaj	25	m	56	5430	gynaecomastia	1	72	28	0.8	134	3.7
10	thavamani	36	f	50	7120	mng thyroid	1	76	30	0.7	136	3.4
11	meena	33	f	52	6091	ca breast	1	78	30	1	135	3.3
12	pavithra	45	f	52	5640	cholelithiasis	1	86	34	8	137	3.5
13	vasuki	36	f	48	8012	cholelithiasis	2	72	20	0.9	138	3.5
14	durai	49	m	56	5098	rif mass	1	88	28	1	135	3.6
15	malliga	51	f	50	6420	ca breast	2	92	27	1.1	136	3.7
16	jeya	53	f	52	7320	ca breast	2	98	24	1	137	3.8
17	keasavan	34	m	56	8006	gynaecomastia	2	112	32	0.9	138	3.8
18	mookan	55	m	54	6408	epigatric hernia	1	102	30	0.8	133	3.7
19	madhan	44	m	52	7054	cholelithiasis	1	98	20	0.8	134	3.6
20	karuppu	46	m	58	7508	cholelithiasis	2	112	20	0.7	135	3.6
21	kani	36	f	52	6987	mng thyroid	1	118	28	0.9	136	3.5
22	aruna	25	f	48	8240	fibroadenoma	2	104	26	0.8	137	3.5
23	karpagam	43	f	50	6726	b/l cervical adenitis	2	108	24	0.9	138	3.3
24	kailash	23	m	56	5480	gynaecomastia	1	76	22	0.7	139	3.7
25	kamala	39	f	50	6087	mng thyroid	2	68	32	0.7	134	3.6
26	manikandan	31	m	54	5624	cholelithiasis	1	70	32	0.8	134	4.2
27	periyanan	50	m	54	7410	epigatric hernia	2	92	34	0.9	136	4.1
28	sundar	36	m	52	5241	cholelithiasis	1	90	20	0.9	134	3.5
29	kannan	27	m	58	5439	gynaecomastia	1	100	24	1	133	3.5
30	pankajam	43	f	50	6552	ca breast	1	102	22	1	137	3.6

GROUP - S (SEVOFLURANE GROUP)

baseline PR	baseline MAP	intraop PR	intraop MAP	No.of Additional doses of Fentanyl	duration of Surgery	time to spontaneous motion	time to response to pain	time to extubation	time to recall of name	time to hand grip	time to achieve PARS>10
76	64	72.2	64	1	110mins	6	8	10	11	13	14
72	66	68.8	64	nil	90mins	7	8	11	13	14	16
74	68	72.2	68.3	nil	100mins	6	7	8	10	12	14
78	66	76.8	64.2	nil	100mins	7	8	10	11	13	15
84	64	82.4	62	1	90mins	6	7	9	10	12	14
88	58	88.2	56.6	nil	110mins	8	9	10	11	13	16
68	66	60.6	68.2	nil	120mins	7	8	10	12	14	16
90	64	84.6	60.8	nil	150mins	7	8	10	13	15	17
92	68	90.2	66	2	90mins	7	9	10	12	14	16
94	66	88.6	64.3	nil	100mins	7	9	11	12	14	16
96	70	88.8	67	nil	110mins	6	8	9	11	13	15
88	66	84.2	63.6	nil	120mins	8	9	11	14	16	18
84	58	80.6	58.8	nil	140mins	6	7	8	10	12	14
72	70	68.6	68.6	nil	110mins	7	9	10	12	14	17
76	66	68.8	65	1	95mins	7	9	12	13	15	17
76	64	70.2	63.3	nil	110mins	8	9	11	13	14	17
72	70	70.2	68.4	nil	100mins	8	9	11	13	15	18
78	66	72.6	64	1	120mins	8	11	12	14	17	19
88	68	84.2	65.4	1	140mins	7	8	10	13	15	18
90	64	86.8	62.3	nil	80mins	8	9	11	14	16	19
92	62	88.2	60	1	90mins	7	8	10	12	13	16
96	66	88.8	64	nil	110mins	7	9	11	13	15	17
84	68	80.2	66	nil	100mins	7	8	10	12	14	17
84	62	80.2	60.2	nil	120mins	8	9	12	14	17	19
92	64	86.8	62.2	nil	140mins	7	9	10	12	14	17
84	66	80.8	63.9	1	150mins	7	9	12	13	15	17
88	68	82.6	66	nil	100mins	8	9	11	13	14	17
84	64	78.8	60.8	nil	110mins	7	8	10	12	14	17
90	62	86.8	60.8	1	100mins	9	10	12	14	16	18
96	66	88.8	62	nil	100mins	8	9	11	13	15	18

COMPARISON OF HEMODYNAMIC, EMERGENCE AND RECOVERY CHARACTERISTICS OF SEVOFLURANE WITH DESFLURANE IN GENERAL ANESTHESIA.

Abstract:

Background:

Inhaled volatile anaesthetics remain the most widely used drugs for maintenance of general anaesthesia because of their ease of administration and predictable intraoperative and recovery characteristics. Management of hemodynamic stability and early recovery is the most important part of a standardized balanced technique. Given the low blood-gas partition coefficients of sevoflurane (0.69) and desflurane (0.42), a more rapid emergence from anaesthesia is expected compared with traditional inhalation anaesthetics. This study was undertaken with the aim of prospectively comparing the hemodynamic, emergence and recovery characteristics of sevoflurane with desflurane in general anesthesia.

Methods:

Sixty ASA I and II patients undergoing elective surgical procedures under endotracheal general anesthesia were randomly assigned to receive either sevoflurane 1% or desflurane 3% for maintenance of general

anesthesia after standardised intravenous induction sequence. Measurement of hemodynamics was done before induction and every 5minutes after induction. During the intraoperative period, if mean arterial pressure increases above 20% of preinduction value, an additional dose of fentanyl was given. Hypotension was treated with intravenous fluids and replacement of blood loss. The number of additional doses of fentanyl required was noted. The time of discontinuation of the inhaled anesthetics to spontaneous movement, response to painful stimuli, extubation, recall of name, handgrip and to achieve a post anesthesia recovery score of greater than 10 were measured.

Results:

Both the groups were comparable in terms of age, sex, weight and duration of surgery. The number of additional doses of fentanyl required was more in the desflurane group, ($p<0.0001$). The time to spontaneous movement, response to painful stimuli, extubation, recall of name and handgrip were shorter in the desflurane group, ($p<0.0001$). The time to achieve recovery score of greater than 10 was shorter in desflurane group with a p value < 0.0001.

Conclusion:

In conclusion, desflurane provides earlier emergence and recovery from anesthesia compared to sevoflurane. Both desflurane and sevoflurane maintained hemodynamic stability, but to maintain the hemodynamics desflurane needed more number of additional doses of fentanyl.

Key words:

Sevoflurane, desflurane, hemodynamic profile, recovery characteristics.